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THE EPIDEMIOLOGY OF POLIOMYELITIS, WITH SPECIAL REFERENCE TO THE VICTORIAN EPIDEMIC OF 1937-38.[1]

By F. M. BURNET.

(From the Walter and Eliza Hall Institute, Melbourne.)

TOWARD the close of the epidemic of poliomyelitis in Victoria during 1937 and 1938 the National Health and Medical Research Council made available facilities for carrying out experimental investigations on the disease at the Walter and Eliza Hall Institute. The detailed results of this work are in course of publication (Burnet, Jackson and Robertson,[1] Burnet and Jackson,[2] Burnet, Jackson and Robertson,[3] and Burnet, Freeman, Jackson and Lush,[4]), but in view of the general interest in the disease it seemed advisable to attempt a more general account of our results, and to indicate their bearing on the general problems of poliomyelitis epidemiology. There is an enormous volume of epidemiological data concerning poliomyelitis, particularly from American sources, but there is strikingly little agreement amongst authorities as to how the findings should be interpreted. There are, for instance, writers who believe that the disease is derived from an animal reservoir with some biting insect as vector (Lumsden[5]), or that infection results from the ingestion of contaminated water or food (Kling[6]), as well as the great majority who accept the view that it is spread by the respiratory route.

In the discussion which follows the presentation of our experimental results, a good deal of use is made of our experience with two other viruses causing human disease (influenza and *herpes simplex*). In our opinion there are important

[1] Work carried out under grant for poliomyelitis research from the National Health and Medical Research Council.

similarities (and differences) in the behaviour of these three viruses, and we feel that results obtained with herpes and influenza, which can be studied experimentally in much more detail than poliomyelitis, throw a good deal of light on the nature of poliomyelitis infection. An effort has therefore been made to provide a considerably more definite statement of the processes concerned in determining the occurrence of poliomyelitis than most authors have felt justified in making.

The General Character of Poliomyelitis Epidemiology.

Poliomyelitis cases appear in two characteristic ways. In most years an occasional child develops the typical illness, usually during the summer or autumn months, without any evidence of contact, direct or indirect, with another case, nor do secondary cases arise from contact with the patient. In England, poliomyelitis has always maintained this character except for the occurrence of a few small local epidemics, often in schools. In some other countries, notably the Scandinavian countries, north-eastern America, New Zealand and Australia, there have been in addition serious epidemics with a relatively high incidence, and in which careful study shows clear evidence of the spread of infection by patients and carriers. The investigations made by the officers of the State Health Department in the Victorian epidemic show this spread from an initial centre of infection very clearly.

The first cases of the epidemic occurred amongst children attending a school in one of the south-eastern suburbs of Melbourne, and there was strong circumstantial evidence that the virus was introduced into this group by an adult who had contracted a mild infection in New Zealand. "Spot" maps of the distribution of cases at progressive stages of the epidemic (Merrilees[7]) give a very clear picture of the way infection spread first in the immediate neighbourhood, then with the appearance of isolated cases in distant suburbs, which in many cases served as centres for fresh spread. Close investigation of the circumstances of these first cases in fresh areas almost without exception allowed the probable chain of human contact to be worked out. Direct patient-to-patient contact was not so common as for the intermediary to be a child with a very minor illness (abortive poliomyelitis). Closure of schools and limitation of the movements of children under sixteen years probably slowed down the spread of the infection to a considerable extent. There was no restriction on the movement of adults, and one can be reasonably certain that carriage of infection by adults was not an important factor in this epidemic. All who were concerned in the search for sources of infection were unanimous that the only likely mode of transference of the virus was by droplet infection, some of the probable infective contacts being only for a very short period. As far as one can gather from the literature, the Victorian epidemic did not differ in any essential respect from recent severe epidemics in other countries.

In epidemic periods, such as one we have experienced, there cannot be the slightest doubt that the infection is spread by contact of susceptible children with infected persons, most frequently with other children in the early stage of a non-paralytic infection. If we accept the view that poliomyelitis is spread in this way, the chief problems which arise, and for which an answer must be sought, are as follows.

1. Why does the behaviour of the disease differ so markedly at epidemic and inter-epidemic periods?
2. What determines whether a child exposed to infection contracts a paralytic or a non-paralytic attack, or avoids infection altogether?
3. What is responsible for the characteristic age incidence of the disease, and for the changes in age incidence in the last forty or fifty years?
4. What part do acquired immunity and the presence of circulating antibody play?
5. Is there any method by which individual protection can be given to exposed children?
6. Can public health measures designed to limit the aggregation and dispersal of susceptibles be effective in controlling epidemics?

At the time our investigations commenced we considered that the most likely answers to these problems could be based on the current American conception of the disease. This conception, which was derived very largely from experimental studies in the monkey (*Macacus rhesus*), may be stated briefly as follows.

The virus of poliomyelitis is a strict neurotrope; that is, it is incapable of multiplying in or damaging any cells other than those of the nervous system. The only point in the body where naked nerve cell protoplasm is on a surface exposed to the environment is in the olfactory mucosa, where the terminal fibrils of the olfactory nerve cells lie free in a film of mucus. This region is the only "natural" route by which the rhesus monkey can be infected with poliomyelitis, and by analogy it is also the usual site of human infection.

The virus reaches the olfactory area by inhalation of infective droplets of saliva or the dry flakes which result from droplet evaporation. Infection of the olfactory cells results, which in a proportion of children spreads into the brain, and by axonal pathways eventually reaches the spinal cord. In a majority of children, however, the infection involves only the olfactory sense cells and perhaps the cells of the olfactory bulb. The symptoms of abortive or non-paralytic poliomyelitis appear, and virus liberated from the infected olfactory cells passes into the naso-pharynx and is eventually set free into the environment with droplets of saliva.

The proportion of paralytic to non-paralytic infections varies from year to year, being highest at epidemic periods. During childhood almost all individuals are infected, but since most of this infection occurs at non-epidemic periods, only a very small proportion become paralysed. The evidence for this widespread subclinical infection is (*a*) the

presence of poliomyelitis antibody in 70% or 80% of normal adult sera, (*b*) the rapid decrease in susceptibility of children with age.

The Significance of Poliomyelitis Antibody in Human Beings.

When we were faced with the problem of deciding what would be the most useful lines of experimental work in connexion with the Victorian epidemic, we decided that there was as yet no real evidence that the passage of an epidemic over a community did in fact result in a large number of subclinical but antibody-producing infections. If the current theory was correct, children attacked with poliomyelitis should be those without antibody, and they should develop it after infection, while amongst family contacts of poliomyelitis patients there should be a high proportion who develop antibody but not paralysis.

Samples of serum were therefore collected from two sources: (1) patients soon after their admission with clinical poliomyelitis to the Queen's Memorial Infectious Diseases Hospital, Fairfield, and (2) from brothers and sisters of several poliomyelitis patients as soon as possible after the diagnosis of the clinical case had been made. One of these contacts subsequently developed paralysis. Blood was taken again from the same patients and contacts two to three months later, that is, at a time when antibody, if it had been produced, should be about its highest level. These samples of serum were tested for their content of poliomyelitis antibody by monkey inoculation.

The method of testing differed from that usually employed in the use of the intraocular rather than the intracerebral route of injection. Preliminary experiments had shown that the intraocular method had important advantages for the purpose of determining whether two sera had or had not the same content of antibody, since it gave more regular results and a higher apparent antibody titre. The results of comparing first and second serum samples from fourteen patients and fifteen contacts were unexpected but perfectly definite. In no case was there any significant change in antibody content resulting from exposure or infection a month or two previously. Further, five of the patients with clinical paralysis had possessed relatively large amounts of antibody in their blood at the time of their acute illness, and equally therefore at the time of their infection a few days before.

These results confirmed those obtained by American workers (Brodie *et alii*[8]) a year earlier, and taken with these demand a considerable reorientation of opinion in regard to the significance of "poliomyelitis antibody". In the first place, it is evident that the possession of circulating antibody is not a guarantee against paralytic infection. Further, if natural antibody has no protective power, it is obviously futile to expect results from the administration of small amounts of human serum, which may or may not contain antibody, to patients with early infections, and it is easy to understand why all controlled series of such therapeutic administrations in recent years have failed to show any benefit. It was of interest to find that when two pooled batches of convalescent serum prepared from early patients in the 1937-1938 epidemic for possible therapeutic use were titrated, one, batch 81, from six individuals, showed a high content of antibody; the other, batch 82, from one adult and one child, possessed practically no inactivating effect whatever for the virus. A careful titration of this last serum was made for activity against a strain of virus isolated from a patient who had died during the epidemic, as well as against our standard "M.V." strain, and only the same equivocal trace of activity was found.

Apart from destroying the theoretical basis for serum therapy, these negative results leave the problem of the origin of the poliomyelitis antibody of normal adults in a very interesting position. If the antibody is not produced during the course of epidemics, where does it come from?

Differences between Poliomyelitis Strains.

When a strain of poliomyelitis virus is first isolated by inoculation of human spinal cord emulsions into monkeys, it usually shows irregularities of behaviour, killing some monkeys and producing only a febrile reaction with little or no paralysis in others. If the virus is transferred from monkey to monkey by intracerebral injection for a long series of animals, it becomes more virulent and will often eventually give almost 100% of fatal paralytic infections. Such a strain is the standard Rockefeller Institute "M.V." strain, which, like many other workers, we have used for most of our serum neutralization experiments. The use of what may be called a "fixed" monkey virus of this sort, although it allows one to obtain very regular experimental results, may have disadvantages. We can be by no means certain that in the course of developing a high virulence for monkeys such strains have not lost essential characteristics of importance for understanding the pathogenesis of human infections.

In the course of our work we obtained the spinal cord from a child, "Mar.", who died of poliomyelitis in Launceston. This, on intracerebral injection into rhesus monkeys, produced typical paralysis of the usual irregular type. With another species of monkey, *Macacus cynomolgus,* from the Malay Peninsula region, however, the original cord material gave invariably fatal paralysis. What was even more striking was that, in this species, intradermal injection of a single dose of "Mar." virus either in the form of the original human material or of spinal cord from infected monkeys, resulted in nearly every animal tested becoming fatally paralysed. The same local strain of virus was also capable of this spread from the skin to the central nervous system of the more resistant rhesus monkeys, although the paralysis produced was rarely fatal. The fixed "M.V." strain, however, failed to infect cynomolgus monkeys by intradermal injection, and many experiments by Rhoads and others had shown that

infection in rhesus monkeys by this route was very irregular. Here, therefore, there is a well-defined difference between two virus strains, possibly related to the fact that one is a recently isolated strain, the other a strain that has had hundreds of passages through monkeys. It is self-evident that the ease with which a poliomyelitis virus spreads from the periphery to the spinal cord may have a very important bearing on its effect in human beings.

Experimental Infection from the Pharyngeal Region.

It was noted by clinicians during the epidemic that there seemed to be an undue proportion of cases in which the primary symptoms were those of paralysis of medullary centres, and it was suggested that these were due to direct spread of the virus from the pharyngeal region along the lower cranial nerves to the medulla. This led us to test the effect of swabbing virus over the pharyngeal region of cynomolgus monkeys.

A total of eight monkeys have been specifically infected by this route, five of them with initial symptoms suggestive of a primary attack on the medullary centres. In two, detailed histological study supported this interpretation. The other three showed primary paralysis corresponding to infection of the lumbar cord, and it is probable that in these infection took place via the bowel. At least an equal number of monkeys, some of which were swabbed over the tonsillar region on several occasions, failed to become infected or died from non-specific causes. Three other cynomolgus monkeys were infected by the gastro-intestinal route, one by inoculation directly into the bowel after laparotomy, and two by feeding.

Previous experiments by Flexner,[9] in which arge amounts of "M.V." virus were fed to cynomolgus monkeys, had given results which were almost wholly negative. The difference in the results can most probably be accounted for by the different viruses used, "M.V." being in our experience unsuited to traverse long nerve paths from the periphery to the central nervous system.

We concluded that our results supported those of Kling, Levaditi and Hornus[10] in demonstrating that cynomolgus monkeys were susceptible to infection by the gastro-intestinal route, using that to signify that infection can occur through unbroken mucosal surfaces somewhere between the pharynx and the lower bowel.

Is there any Evidence that the Virus Usually Causes Infection via the Olfactory Nerves?

The most recent attempt to diminish the incidence of poliomyelitis amongst potentially susceptible children has been by application of zinc sulphate solutions to the olfactory mucous membranes. This procedure is based entirely on experimental findings in rhesus monkeys. These animals can be infected with almost complete regularity if a large dose of virus is dropped into the nose while the monkey is held in a partially inverted position, and they can be rendered resistant to infection by application of various protein coagulants to the olfactory mucosa. Zinc sulphate is the most effective of these, a single application of a 1% solution giving a resistance to infection which lasts at least three months.

In the rhesus monkey there is no doubt that infection by this route passes to the central nervous system by the olfactory nerves since the virus can be isolated from the olfactory bulbs forty-eight hours after the nasal instillation, and histological section of the bulbs shows characteristic changes not seen when the monkey is infected by other routes (Sabin and Olitsky[11][12]). As I have already mentioned, however, cynomolgus monkeys can be infected by natural methods which do not involve the olfactory region, namely, feeding or pharyngeal swabbing. Cynomolgus monkeys are definitely more susceptible to infection by our recently isolated strain than are rhesus monkeys, and since the poliomyelitis virus is certainly more highly infectious for susceptible human beings than for either species, it is probably justifiable to think that the action of the virus on the more easily infected species (cynomolgus) would more closely resemble its action in children. In the circumstances, the fact that the olfactory route is the only "natural" route by which rhesus monkeys can be infected has no necessary bearing on the pathway by which human infection results. The pathogenesis of the human infections can be worked out only on human material. Using material from the Victorian epidemic, Robertson[13] has studied the histology of olfactory bulbs removed from children who had died of poliomyelitis. Of the eleven bulbs examined only one showed any pathological change (perivascular cuffing). This negative result can be interpreted in one of two ways. Either the virus passes through the human olfactory bulb without producing the lesions seen in monkeys, or it usually reaches the central nervous system by some other route. There is probably no satisfactory basis for a decision between the two alternatives, but at least the results give no positive support for the hypothesis of olfactory entry.

As far as can be determined from the reports of field tests on the efficacy of zinc sulphate spraying, the results have shown no significant benefit (Tisdall[14] and others). There are, however, very great technical difficulties in the carrying out of such field experiments with an adequate control series and on a scale large enough to provide statistically useful results.

On the whole, the available evidence is rather opposed to the view that the olfactory route is the common portal of entry for the virus in an epidemic. If positive evidence could be obtained of infection by other routes there could be no hesitation in abandoning the olfactory hypothesis.

Age Incidence of Poliomyelitis.

The preceding three sections cover the essential result of our own investigations. If we are to bring these into their relation with general conceptions of the natural history of poliomyelitis, it is necessary to consider some of the more strictly epidemiological aspects, particularly the age incidence of the disease. From many points of view the distribution

of cases according to age is the most useful source of information available for understanding the behaviour of an infectious disease. It has the great advantage of being a strictly objective finding, easily obtained by a simple inspection of records, and therefore free of the possible errors and misinterpretations which are inevitable in attempts to elucidate such matters as incubation period, exposure to infection and past history by the study of individual cases. Under standard conditions the age incidence graph of any infectious disease has a characteristic shape, and any significant change from this shape will signify some change either in the pathogenic microorganism or in the population exposed to infection, and will usually give some indication of the factor responsible.

There is a great deal of statistical material available in regard to the age incidence of poliomyelitis in different human communities and at different times, and we may summarize this data under several headings:

1. Confining ourselves for the present to poliomyelitis as it appears in large towns and cities in European, North American or Australasian communities, the first thing to note is the great change in age incidence which has taken place since the early years of this century. In practically all the epidemics described before 1910 the incidence was predominantly in infants or very young children—hence the name infantile paralysis. The classical Swedish epidemic of 1887, from which Medin first clearly deduced the infectious character of the disease, involved 44 children, of whom 35 were under three years of age. In Figure I are collected age incidence graphs of three early epidemics in cities of Australia, Sweden and United States of America. All show an essentially similar character, with from 60% to 90% of the cases falling in the age group nil to five years.

When we compare these graphs with others representing the age incidence of cases occurring in the same countries in epidemics subsequent to 1930, a very marked and uniform change is evident (Figure I). With few exceptions, the age of maximum susceptibility has risen, so that in most communities there are now at least as many cases in the age group five to ten years as from nil to five years. In several countries recent epidemics have shown relatively large numbers of adult patients. The graph for the epidemic in Eskilstuna (Sweden) 1935-37 shows one example of this. Others may be found in the Californian experience in 1934, where 37% of the cases reported were in persons over 15 years of age, and in Denmark 1933-34[15], where 33·2% of the patients were over 15 years of age. To a certain extent this rise in age incidence is due to the recognition and reporting of non-paralytic infections, but the age incidence of deaths has shown a corresponding change, so that a real change in the behaviour of the disease has evidently occurred. It should be noted that at least two epidemics in recent years — Freistadt, Germany, 1927,[16] and Alsace, 1930[15]—have shown the old type of age incidence, with about 80% of patients under five years of age.

2. In the period prior to 1920 the peak of age incidence falls at a higher age in rural communities than in cities. The predominantly rural State of

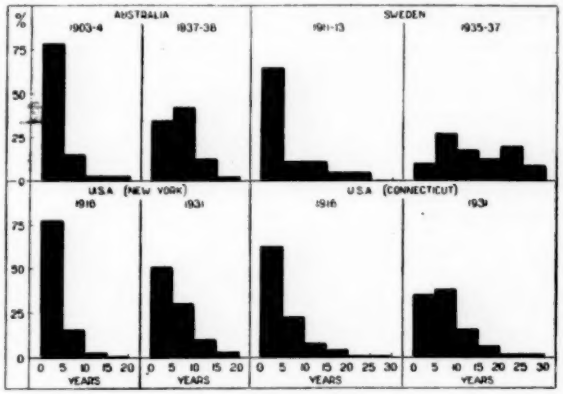

FIGURE I.

Age incidence of poliomyelitis in urban populations to show the change which has taken place since about 1918. The graphs show the percentage of total cases reported which fall in successive five-year age groups. *Australia*: (1) Epidemic of 1903-4 in Sydney and Brisbane. Combined figures from Wade,[23] Litchfield[24] and Ham.[25] 167 cases. (2) Victorian epidemic of 1937-38. Melbourne figures from State Health Department. 1,023 cases. *Sweden*: (1) Epidemics of Stockholm, Malmo and Gothenburg, 1911-13, from Jönsson.[26] 434 cases. (2) Eskilstuna epidemic, 1935-37, from Jönsson.[26] 114 cases. *New York City*: (1) Epidemic of 1916. 9,345 cases. (2) Epidemic of 1931. 4,100 cases. Figures from "Poliomyelitis".[16] *Connecticut*: (1) Epidemic of 1916. 856 cases. (2) Epidemic of 1931. 1,133 cases. Figures from "Poliomyelitis".[16]

Vermont (United States of America) may be taken as an example. Even from the earliest reports (1894) there was a notably high proportion of older patients, and an analysis of the age incidence in different types of community in Vermont over the period 1912-1925 showed a consistent relationship between density of population and the age incidence of cases. These figures, from the Milbank Monograph[16] are reproduced as Table I, which also includes for comparison the corresponding figures for the New York epidemic of 1916. Such differences

TABLE I.

Variation of Age Incidence with Density of Population in North-Eastern United States, 1912-1925.[16]

Centre of Population.	Age Group.			
	0 to 5 Years.	5 to 10 Years.	10 to 15 Years.	Over 15 Years.
New York City (1916)	78·7	16·8	2·5	2·0
Vermont cities ..	60·4	20·8	7·8	11·0
Vermont towns and villages ..	36·2	30·8	11·3	21·7
Vermont isolated farms	24·2	25·7	18·9	31·1

between town and country appear now to be much less evident than formerly. As was noted above, the difference in age incidence between city and country in the Victorian 1937-1938 epidemic was very slight, the only notable feature being that cases

in persons over fifteen years of age were about twice as frequent in the country as in the city, but in both groups these made up a very small proportion of the whole.

3. The age incidence has an earlier peak in warm than in cold climates, if the difference found by Doull[17] between the northern and southern States of the United States of America can be ascribed to that factor alone. In the northern States 27·7% of the total deaths occurred among infants under two years, while the corresponding southern States' figure was 59·7%. As far as data are available, the difference is due to an increased proportion of cases at these ages in the south rather than to an undue fatality.

4. During the course of an epidemic the peak of age incidence tends to rise. This has been noted in the New York 1916 epidemic,[16] Ohio, 1927,[16] and also in the recent Victorian epidemic.

5. There is little or no evidence that infants possess passive maternal immunity during the first months of life. The incidence in infants aged under one year in New York in 1916 was twenty-five times that in adults, who must have been much more exposed to infection; and there were many cases in children under six months.[16]

6. All the available evidence suggests that the virus of poliomyelitis is widespread throughout the tropics as well as in temperate regions, but there are several epidemics on record in which "virgin soil" communities appear to have been affected. These include four Pacific island epidemics[16] and one in northern Greenland (Høygaard[18]). In all, the major incidence was on the young adult population. In contrast to the north Greenland epidemic, is one reported from a settlement in the south, Sukkertoppen.[15] This suffered an epidemic in 1914 and again in 1932. On the second occasion a very high proportion of the children were infected (83 of a total population of 700 individuals of all ages), but persons born before 1914 were unaffected.

In any attempt to provide an interpretation of these findings and to bring them into relation with the properties of the virus and the clinical character of poliomyelitis, it is necessary first to separate the experience of earlier years from that subsequent to 1920. For the earlier period we may take the experience of the north-eastern States of the United States of America, culminating in the great New York epidemic of 1916, as providing the most complete material.

1. The first point of importance is the peak of incidence at the earliest ages (0 to 3 years). There is only one other specific infectious disease which shows this character, *herpes simplex*. Burnet and Williams[19] have shown that infection with this virus commonly occurs before the age of three years, and, confirming Dodd, Johnson and Buddingh,[20] find that the characteristic primary infection takes the form of aphthous stomatitis. Infection is obviously from the parents, 90% to 95% of whom are infected with the virus and liberate it in the course of recurrent attacks of *herpes labialis*. By contrast, infections such as measles, chickenpox and diphtheria, which are spread by contact between children, show a peak at early school age. The obvious inference is that poliomyelitis was due to infection from adult carriers of the virus, particularly the parents of the children infected.

2. The difference in the age incidence of city and country can be accounted for only if we make several important assumptions. (*a*) That in non-epidemic years in New York, for example, young children are infected to almost the same extent as they are in epidemic periods, since there is no indication from available figures that the period since the last epidemic in endemic areas influences the age graph, but that the great majority escape clinical paralysis and are subsequently immune to the paralytic action of the virus, though not necessarily to infection. (*b*) It follows from this that most adults must at some time in each year be carriers of the virus, otherwise the young children would not obtain that immunity which is shown to some extent even by three-year-old and four-year-old children in epidemic periods. Either, as in the case of herpes, a big proportion of adults are permanently infected with the virus and liberate it at intervals, or a similarly large percentage are intermittently infected, possibly in the pharynx, by whatever strains of poliomyelitis virus are circulating in the community. Since the morbidity from poliomyelitis varies greatly from year to year, we are probably compelled to adopt the second of these alternatives. (*c*) In rural areas there is less active dispersal of respiratory infections, and both immunizing and paralytic infections are liable to reach the child at a later date than in the city.

The occurrence of adult carriage of the virus is generally recognized, but it has rarely been given the importance that has been ascribed to it here, and most laboratory workers would certainly dissent from the opinion that recurrent superficial pharyngeal infections by any type of poliomyelitis virus could occur. Their objections would be: (i) that poliomyelitis virus in the only animal available for experiment is a strict neurotrope, and cannot therefore be expected to produce superficial mucosal infections in man; (ii) that recurrent superficial infections are unlikely to occur in virus diseases owing to the establishment of immunity after the first infection. The reply to these objections is: (i) The behaviour of a virus in its normal host and in a laboratory animal may be very different. According to Hurst,[21] pseudorabies virus in the monkey is a strict neurotrope, producing always fatal infections of the central nervous system. In its natural host, the pig, a mild superficial infection of the upper respiratory tract is produced, and contact infection readily occurs. (ii) In *herpes simplex* recurrent infections (of endogenous origin) are almost invariable, despite the presence of a large amount of circulating antibody. A closer parallel to the hypothetical conditions in polio-

myelitis can be found in recent unpublished work by Burnet, Lush and Cade on influenza virus infections. In a community heavily exposed to infection, a very high proportion of subclinical infections occurred in adults, many of whom had substantial initial amounts of antibody in their serum. The occurrence of subclinical infection was deduced from the observation of a sharp rise in virus-inactivating antibody titre.

Finally, there is the experimental fact that virus may be obtained with fair regularity in tonsils removed from patients in the acute stage of the disease, and in one instance (Kramer[22]) from a healthy adult, or from the naso-pharyngeal washings in paralytic or abortive cases. When one considers the difficulty of establishing the presence of virus in many specimens of human spinal cord removed *post mortem*, the fact that it can be detected in washings from the naso-pharynx, in fæces or in excised tonsils, suggests strongly that larger amounts are present in the pharynx than could be accounted for by leakage from the olfactory cells. Several European authors have stated that poliomyelitis is more liable to affect children of adults whose duties bring them into contact with large numbers of persons, and in accounts of epidemics in extremely isolated communities it is almost the rule to find the initiation of infection traced to a healthy adult arriving from some place where the disease is endemic or epidemic. One must conclude that infections with poliomyelitis virus of pre-1920 type were frequent and recurrent in adults in much the same way that streptococcal infections are. Further, it appears almost certain that the infection was a pharyngeal one rather than intestinal, and the mode of transfer droplet infection.

3. The incidence at earlier ages, which is found in warmer climates, is probably to be correlated with the much lower general incidence of paralytic poliomyelitis there. As is well known, diphtheria is rare in the tropics, yet the early appearance of "negative" Schick reactions in almost every tropical population tested makes it certain that subclinical infection is widespread. In precisely similar fashion poliomyelitis virus is also widespread, and infection occurs in most children at an early age; but a much larger proportion of the infections are subclinical than in colder climates. The concentration of paralytic infections in the lowest age group may be merely an expression of the fact that all older individuals have been immunized; but, as will be discussed later, there is also the possibility that physiological factors allow an easier spread of virus from the periphery to the central nervous system in the lower age groups. Apart from the analogy with diphtheria and streptococcal infections, there is no indication why the proportion of subclinical infections should be higher in the warmer climate.

The position, then, in regard to poliomyelitis before 1920 is reasonably intelligible on the hypothesis of widespread and recurrent infection of both adults and children by a virus normally of low virulence but high infectivity. Such subclinical infections provide a substantial immunity against the paralytic effect of subsequent contact with an unduly neurotropic strain, but not against recurrent superficial infection, presumably of the pharyngeal mucosa. It is reasonable to assume that circulating antibody was produced by these recurrent infections of non-nervous tissues.

Passing now to epidemic poliomyelitis as we observe it at the present time, and taking the 1937-1938 Victorian epidemic as a typical example, we find a considerable difference from conditions in the earlier period. The incidence of the disease now resembles much more closely that of measles and diphtheria, and, like these, it is quite clearly spread by child contact, and only exceptionally by adult carriers of the virus. In the Melbourne epidemic, despite free movement of adults, nearly all the early spread from one suburb to another could be ascribed to child contacts with a fair degree of certainty (Merrilees[7]). Infants were not often infected, except from older children in the household. As has been pointed out by several authors, urban poliomyelitis now resembles closely rural poliomyelitis of the earlier period. In Victoria there was only a very slight difference in the age incidence between city and country in 1937-1938.

The difference between the two periods may clearly be a result of change either in the virus or in the community exposed to its activity, or in both. On the whole we can feel certain that changes have occurred in the character of the dominant virus strains at different periods and also that changes in the human environment have been largely responsible for these changes. It appears that throughout its history as a producer of epidemic disease—that is, from about 1880—poliomyelitis virus infection has shown a general tendency to change in the direction of a greater neurotropism. We can tabulate the differences which may be taken to exist between the virus responsible for the very low incidence of sporadic cases in the period before 1887 and such a virus as was responsible for the Victorian epidemic of 1937-1938 as follows:

Virus of Endemic Poliomyelitis of Nineteenth and Early Twentieth Centuries.	*Virus of Recent Epidemic Poliomyelitis.*
Essentially epitheliotropic, probably infecting mainly pharyngeal mucosa; infection of central nervous system a rare accident.	Much more neurotropic, passing readily to the central nervous system.
Recurrent non-symptomatic infection common, so that adults are frequent carriers and liberators of virus.	Non-symptomatic infection of adults rare, and spread almost entirely by contact of children.
Poliomyelitis antibody produced by infection, particularly when recurrent.	Poliomyelitis antibody not produced by a single infection with the virus.
Infection normally provides a degree of immunity which will protect against paralytic infection by epidemic virus of the pre-1918 type, but only to a much more limited extent against the recent epidemic type.	Temporary immunity only is produced.

It must be understood that these two types of virus represent the extremes of a continuous range. The virus of the New York 1916 epidemic, for

instance, must have retained the power to produce many non-symptomatic infections in adults, but had developed a high power of spread to the central nervous system in those children not protected by previous subclinical infection. The recent epidemic type has become a more specialized parasite, and has to a large extent lost the power to produce the superficial adult infections characteristic of the earlier strains. The assumption that strains of the earlier type have persisted, but that superimposed on their activity we have had epidemics due to progressively more specialized neurotropic strains, provides the simplest interpretation of the epidemiological facts.

Ecological Considerations.

So far we have considered the problems of poliomyelitis almost wholly from the human point of view. It is always valuable to look at the problems of an infectious disease also from the point of view of the microorganisms responsible. An infectious disease persists only when the microorganism is fitted to survive indefinitely in the particular form of parasitic existence to which it has become adapted. In general, a stabilized parasite-host relationship is indicated by a high proportion of infections and a very low mortality. A high mortality in the host is usually a factor diminishing rather than facilitating the survival of the parasite species responsible, and most epidemiologists consider that a period of intense activity of an infectious disease indicates always a transitional unstable phase following on some important change in the ecological relationships of host and parasite.

Only in the case of rabies is there any indication that the neurotropism of a virus is a character of adaptive survival value. In poliomyelitis the passage of virus to the central nervous system with paralysis or death of the host can obviously do nothing to facilitate its transfer to another susceptible individual. The neurotropism of poliomyelitis virus must be a simple biological accident, and all the indications are that in the pre-epidemic era the virus had developed a satisfactory mode of existence as a mild parasite of the human pharyngeal region, its latent neurotropism becoming manifest only on rare occasions. For reasons which I have given earlier, the standard form of infection produced by the virus in the pre-epidemic era is presumed to have been a superficial infection, probably of the pharynx in adults. Primary infection occurred nearly always in infancy, and must therefore have been derived as a rule from frequently recurrent infections in parents or older children.

A hint as to why the virus began to develop its modern epidemic tendency about the beginning of this century may perhaps be found in the fact that the countries which have suffered most severely, though by no means exclusively, from poliomyelitis epidemics, are those with the highest standard of living and of personal hygiene. The Scandinavian countries in Europe, north-eastern America, Australia and New Zealand have the lowest general death rates and the worst poliomyelitis histories in the world. The suggestion arises that the change in character of poliomyelitis is related to the effect of improved social conditions in diminishing the ease of spread of respiratory infections. Direct evidence that social and economic changes influence the spread of infection in childhood can be obtained from two sources. Recent surveys of university students and similar groups in the United States of America have shown that the proportion of young adults with evidence of past subclinical infection by diphtheria and tubercle bacilli has diminished considerably in the past twenty years. Our own work on the distribution of herpes antibody in different social groups in Melbourne showed that persons living in more prosperous circumstances had avoided infection with the virus much more successfully than those from working-class areas. Just what factor associated with better economic status resulted in this difference is unknown, but it is reasonable to assume that similar factors would tend to reduce the proportion of infantile poliomyelitis virus infections in an economically improved community. It seems that for the virus to maintain its original character and its equilibrium with the host species, the standard mode of transfer must be from one semi-immune adult to another, primary infections of children accounting for only a small proportion of transfers. When social factors diminish the extent to which both adult and infantile infection occurs, relatively large groups of susceptible children of school age may accumulate in a way impossible under the older conditions. It now becomes possible for the virus to undergo a series of passages from the tissues of one non-immune child to another, and it is more than probable that passage in this way through a long series of primary infections in children would alter the character of the virus in the required direction.

From the point of view of the virus as a species, when the possibility becomes available of maintaining its existence by rapid passage through uniformly susceptible hosts, there is no longer the necessity for the virus to retain the capacity to infect partially immune adults. It is a general rule that the adoption of a specialized parasitic role by an organism results in a loss of its former ability to survive in a saprophytic or semiparasitic fashion. The greater neurotropism of recent poliomyelitis strains is still to be regarded as an essentially accidental character, but one to be correlated with the diminution in the power of the virus to produce adult infections, which has resulted from its ability under modern civilized conditions to find an easier mode of living by transfer from child to child.

Immunity in Relation to Recent Epidemic Poliomyelitis.

The foregoing discussion on the changes in the age incidence of poliomyelitis can now be brought into more direct relationship with recent experi-

mental work on the significance of poliomyelitis antibody in human beings. It has been shown that a single infection with recent epidemic virus does not provoke the appearance of antibody in those not previously possessing it, and, further, that a proportion of children with antibody at the time of exposure nevertheless contract paralytic poliomyelitis. Brodie, Fischer and Stillermann's[8] work indicates also that the proportion of paralytic cases with antibody is lower than is found in a normal population of the same age, but that amongst children diagnosed as suffering from non-paralytic poliomyelitis the proportion with antibody is essentially the same as amongst normal children.

As we have discussed elsewhere (Burnet and Jackson[2]), there are three possible origins of the antibody to be considered.

1. It is a result of normal physiological processes, a "maturation" antibody whose specific relationship to poliomyelitis virus is essentially fortuitous. The surface structure of the adult antibody globulin is assumed to be such that the determinant groups of the virus are specifically bound to it, while the globulin of some young children has no such capacity. This hypothesis can be eliminated at once if we consider the facts that some children under five have antibody, while some adults have none. The distribution of antibody according to age is what would be expected if its appearance was determined by some commonly encountered but not inevitable environmental stimulus, of which infection by a microorganism would be by far the most likely.

2. The antibody is a response to an antigenic stimulus by a microorganismal invader, either (*a*) a virus antigenically similar or identical with poliomyelitis virus, but with other characteristics than that of recent epidemic poliomyelitis, or (*b*) some unrelated microorganism which quite fortuitously possesses antigenic determinant groups in common with the virus antigen.

The assumption that poliomyelitis antibody is produced as a response to some quite different microorganism is not intrinsically absurd in view of the Weil-Felix reaction in typhus fever, but it could be seriously considered only if some direct experimental evidence were available, or if no other alternative offered The most natural hypothesis is that the antibody results from infection with strains of poliomyelitis virus which differ from the epidemic variety in some way that makes them more capable of providing an adequate antigenic stimulus. The absence of antibody production in children with paralytic infections by recent strains of virus can be reasonably ascribed to the predominantly neurotropic character of the virus. Apart from the initial superficial infection, the proliferation of the virus takes place entirely in nervous tissue, and the virus does not reach the antibody-producing cells of the body in sufficient amount to provide an adequate stimulus.

In view of the change in character of the virus which must be postulated to explain the changed age incidence of the disease, it does not necessarily follow that all strains of virus would resemble the current epidemic ones in this respect. In fact, the characters we have ascribed to the pre-1918 strains of poliomyelitis to account for their epidemiological characteristics are just such as would make them much more potentially effective as antibody producers than recent epidemic strains. If we accept the presence of poliomyelitis antibody in human serum as evidence of infection with the virus, we are forced to postulate the continued existence of poliomyelitis virus strains of what we have called pre-1918 type, that is, strains which are capable of producing recurrent pharyngeal infections with only a very slight tendency to involve the central nervous system.

Judging from the figures by Brodie, Fischer and Stillermann,[8] subclinical infection by antibody-producing strains has some influence in diminishing the likelihood of subsequent paralytic infection by epidemic strains, but this relative immunity is much less potent than against the former type of epidemic. Resistance to epidemic poliomyelitis is probably to a large extent non-specific in character. It will be remembered that the virus responsible is, unlike the earlier strains, assumed to be much less readily capable of producing recurrent pharyngeal infections in adults. Looked at from a different point of view, this is to say that with increasing age children develop a non-specific resistance to superficial infection with the virus. Such a development of resistance finds a close analogy in the behaviour of the body toward *herpes simplex* virus. If primary infection is avoided in early life, the individual remains free from herpetic infection indefinitely, although in a city he must be exposed to infection on numberless occasions. Superficial infection (stomatitis) appears to be possible only in the first few years of life, but if it occurs then the virus remains permanently established. The peculiar character of herpes infections makes it possible to be very definite that the resistance to primary infection which develops with age is in no sense of immunological origin. One cannot be so definite in regard to poliomyelitis, in view of the predominantly adult incidence of paralysis in virgin soil epidemics and the appearance of a distinct "young adult" peak in the age incidence graph of some recent Swedish and American epidemics. Nevertheless, there is certainly a very much lower susceptibility to paralytic poliomyelitis amongst the 20% or 30% of adults who possess no poliomyelitis antibody than in a comparable group of five- or six-year-old children.

The Present Position in Regard to the Control of Poliomyelitis.

We may first summarize the general conception of poliomyelitis which we have reached as follows. Poliomyelitis virus strains derive eventually from a virus which, prior to the end of the nineteenth

century, was responsible for frequent infections of some part of the naso-pharyngeal mucosa. On rare occasions primary infection by the virus in unimmunized individuals resulted in spread of the virus to the central nervous system and clinical paralysis. This happened only on first infections, which, owing to the wide diffusion of the virus, could naturally occur only in very young children; hence the characteristic age incidence which gave rise to the name "infantile paralysis". At this period the virus was capable, like influenza virus is today, of producing superficial infections in adults or older children with a basic immunity from former infection. This immunity was, however, sufficient to ensure that spread of the virus to the central nervous system did not follow these recurrent infections.

With the development and dissemination of modern ideas of comfort and cleanliness, opportunity for the free diffusion of the virus became more limited, and a larger proportion of children passed through infancy without being infected. This provided an essentially new means of spread for the virus by contact of susceptible children instead of transfer between partially immune adults. In this new biological environment the virus gradually changed its character, becoming (*a*) more infectious for children, (*b*) less capable of infecting adults, and (*c*) more highly neurotropic. The first two of these changes are the important ones; the increased neurotropism is to be regarded as a rather accidental feature, and one which varies greatly from one prevalence to another. When the neurotropism is high, recent strains appear to be capable of overcoming an immunity which would have provided a solid protection against paralytic infection by strains of the earlier type.

The present position as we see it in a community such as Victoria is as follows.

1. There is probably still a good deal of old type poliomyelitis virus infection, capable of producing antibody and giving recurrent subclinical infection in adults, but this is diminishing, and is not often transferred to infants.

2. The important feature is that the dominant strains are now spread like measles, essentially by child contact. The proportion of paralytic cases varies in different prevalences, but in such an outbreak as the epidemic of 1937-1938 may be very high. A single infection does not provoke the appearance of antibody, and it is by no means certain that immunity to reinfection is more than temporary. Previous infection with an antibody-producing strain does not ensure immunity from paralysis by an epidemic strain.

3. Infection occurs by inhalation or ingestion of contaminated saliva from an infected child, the primary focus produced is in the mucosa probably of the pharyngeal region, and infection may reach the central nervous system by a variety of nerve paths. The olfactory route is probably a rather unusual one.

4. Resistance to paralysis by epidemic strains develops with age, irrespective of specific immunological factors. The general Australian experience is that resistance to paralysis is greater in the warmer regions, and that even a highly virulent strain introduced into a susceptible population, as at Mildura in 1937-1938, may produce a very small proportion of paralysis. Further, there is evidence that an epidemic may cease for as yet unexplained reasons before all the susceptible population has been involved.

On this basis we can consider what are the possibilities of controlling the incidence of the disease. On the whole, the outlook is not promising. Attempts at providing individual protection to exposed children have all failed. Vaccination by partially inactivated virus has proved dangerous, and the use of killed virus would probably be quite ineffective. Passive protection with human serum during epidemics has been tried, but there is very little theoretical basis for its use, and the reported results are not convincing. The attempt to prevent infection by chemical blocking of the olfactory area was based entirely on animal experiments, and in the only controlled series available had no signficant effect in practice. There may well be other ways of inducing resistance to paralytic infection which have yet to be investigated, and it seems likely that a close investigation of the influence of climate and other environmental factors on the course of epidemics might suggest practical measures of individual protection.

The protection of the community by public health measures offers an equally difficult and at present insoluble problem. The only general principle available is to restrict the aggregation of children as much as social considerations will allow, to isolate known child contacts for a period of three weeks, and to enforce a similar period of isolation on all children going from an infected to a non-infected community. Such measures will not stop an epidemic, but they will certainly slow down its spread and almost certainly reduce the total number of paralyses over the whole epidemic. Since epidemics nearly always terminate with the onset of cold weather, a "slowing-down" policy may result in a large number of suceptibles being still untouched when climatic or other non-specific factors terminate the epidemic.

In the past there have been doubts expressed as to whether anything is to be gained by endeavouring to limit the disease by such measures on the ground that it was inevitable that any child would eventually be infected, and that on the whole the younger the age at which primary infection occurred, the less likely was death or paralysis to result. This view cannot be supported if the evidence we have presented that resistance against modern epidemic poliomyelitis may develop irrespective of the occurrence of specific immunization be accepted. In the face of a serious epidemic such as the Victorian one of 1937-38, there is every justification for active

measures being taken to block its spread. Figure II, which shows the number of cases reported each week in six Melbourne districts during the epidemic, is of considerable interest in indicating what can or might be done. The epidemic began in the "bayside" suburbs and in the early stages great efforts to immobilize children in this area were made. When the spread to other suburbs occurred, public alarm had lessened somewhat, and particularly in the industrial suburbs isolation of children was less practicable than in the middle class bayside area. In the eastern suburbs, including all the better

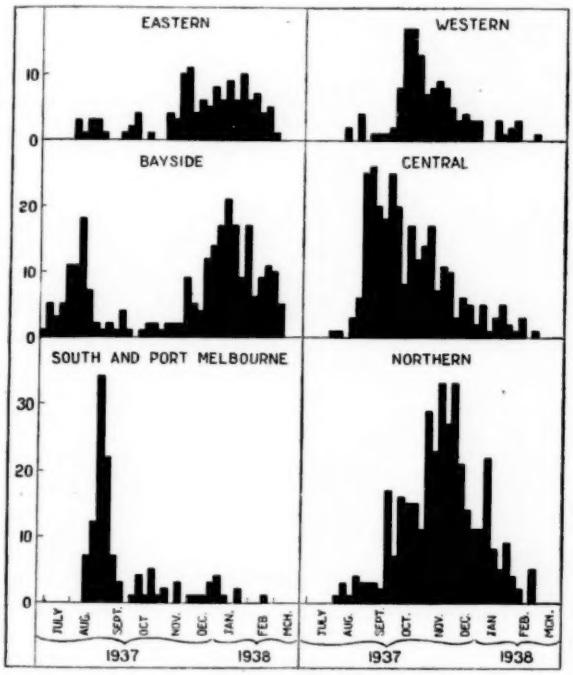

FIGURE II.

Weekly cases reported during the Melbourne epidemic from different areas of the city. *Bayside*: St. Kilda, Caulfield, Oakleigh, Brighton, Sandringham, Moorabbin, Mordialloc and Chelsea (residential). *Eastern*: Kew, Hawthorn, Camberwell, Box Hill, Prahran, Malvern (better class residential). *Western*: Williamstown, Footscray, Essendon and Braybrook (industrial). *Central*: Melbourne, Fitzroy, Collingwood, Richmond (industrial). *Northern*: Brunswick, Coburg, Preston, Northcote, Heidelberg (mainly industrial). *South and Port Melbourne* (industrial).

middle class residential areas, isolation of young children was very general, and facilities for doing so were usually available. From the graphs it will be seen that the bayside region was the only one to suffer two distinct prevalences, the second presumably involving essentially those children who were protected from the first by the strict isolation enforced during the early days of the epidemic. In the industrial suburbs there is a single peak of incidence, while in the better class area the total incidence is low and is spread rather evenly over a long period.

The epidemic stopped rather suddenly in the autumn, at a time when one could feel certain that at least one important country district, the south-west, and one suburban district of Melbourne (eastern) had not been by any means exhausted of susceptibles. The lesson seems to be that during an epidemic severe enough to show evidence of local spread, every effort should be made to prevent infection of children by measures of the sort adopted in the Victorian epidemic.

Summary.

1. A brief account is given of experimental work done in connexion with the Victorian epidemic of 1937-1938.

2. The most important results of this work were to indicate (*a*) that infection in the epidemic did not produce poliomyelitis antibody, (*b*) that certain children with initial antibody nevertheless suffered from paralytic infection, (*c*) that cynomolgus monkeys could be infected with the local virus by ingestion or by pharyngeal swabbing.

3. A discussion of the age incidence of poliomyelitis leads to the conclusion that an important change in the character of the disease has taken place in the last thirty or forty years. The interpretation suggested is that in the earlier period poliomyelitis was essentially an adult infection with incidental involvement of infants, while epidemic poliomyelitis now is a disease of childhood, spread by child contact.

4. The implications of this hypothesis in regard to the measures called for in the control of an epidemic are discussed.

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SOME PROBLEMS OF THE FIRST YEAR OF LIFE AND THEIR MANAGEMENT.[1]

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THE object of this paper is to bring before the notice of the medical profession certain aspects of some diseases which occur in the first year of life, with the hope that treatment may be more efficiently carried out. Artificial feeding has been discussed in connexion with various problems, but more from the aspect of treatment than from that of a discourse on infant feeding.

Hæmorrhagic Disease of the New-Born.

In the treatment of hæmorrhagic disease of the new-born a decision must be made between blood transfusion and the intramuscular injection of blood into the buttocks. The mild case, in which there is a small quantity of blood in the fæces, will undoubtedly respond to the injection of 10 cubic centimetres of blood into each buttock; but it is highly probable that such an infant would survive without this treatment. In the more severe cases, in which pronounced melena and hæmatemesis are present, it is imperative that a blood transfusion should be given. The best procedure is to expose the internal saphenous vein just above the ankle joint, and then, with the assistance of a three-way cock and serum syringe, slowly introduce the blood, which has been correctly typed, in the amount of 10 cubic centimetres per pound of body weight. The blood cannot be given satisfactorily by the gravity method. The administration through the superior longitudinal sinus is much more difficult, and at the same time not without its risk to the infant on account of the danger of extradural hæmatoma or meningeal reactions. If this site is used, then a Kauf-Luer syringe should be used.

The administration of blood into the peritoneal cavity in my experience has been a failure, and must not be used in the hope that the bleeding can be stopped by this means.

In the severe case it is rather disconcerting to see the melena persist for twelve hours after the transfusion; but if sufficient blood has been given early enough, no more is required.

Subdural Hæmatoma in the New-Born.

Although subdural hæmatoma is not a common finding in the new-born, yet my experience of two infants with this complaint in the last eighteen months has prompted me to report these cases, with the hope that others will be diagnosed, and successfully treated.

CASE I.—Ian J., a first baby, was born on July 16 by breech delivery, without difficulty in the delivery of the head. He was seen on the fourth day, July 19, in a fit involving the right side of the face and the right arm and the right leg; the head and eyes were turned to the right. Examination revealed an infant who was drowsy, pale, suffering from shock and definitely icteric; the anterior fontanelle was bulging, the right leg and arm were slightly spastic and bilateral adductor spasm was present. A large bore needle was introduced through the anterior fontanelle into the subdural space on each side, and with a serum syringe 35 cubic centimetres of old blood were removed from the left side and five cubic centimetres from the right side.

On July 20 the child had more fits in the morning; five cubic centimetres of blood were removed from the left side and half a cubic centimetre was removed from the right side. On July 22 there was another fit, and three cubic centimetres were removed from the left side. On July 24 the child had another fit, and 2·5 cubic centimetres were removed from the left side. On July 27 there was no fit, but two cubic centimetres were removed from the left side. On July 31 the anterior fontanelle was full and blood was present in the fæces; 25 cubic centimetres of blood were removed from the left side. On August 2 15 cubic centimetres were removed from the left side. On August 3 the baby was given a transfusion of 90 cubic centimetres of his father's blood. On August 4 one cubic centimetre was removed from the left side. On August 8 the head was fourteen and a quarter inches in circumference; no blood was removed.

Six months later the child's head was sixteen and a half inches in circumference, and he looked quite normal. At four months of age he could support his own head, and there was no sign of spasticity. Six months later again his head was seventeen and a half inches in circumference; the anterior fontanelle was closed, and he had been saying odd words for months. There was no sign of spasticity, and he looked a normal infant. There has been no sign of any fits since the first ten days of life.

CASE II.—Baby F., a male, a first baby, born on September 8 in the anterior position; delivery was instrumental and without difficulty. The child sucked the breast normally in the first two days. On September 11, in the afternoon, he appeared drowsy and refused the

[1] Read at a meeting of the South Australian Branch of the British Medical Association on November 30, 1939, at Adelaide.

breast. On September 12 he refused the breast and vomited on one occasion. The matron reported twitching of his right arm and leg. On September 13 vomiting was frequent; pronounced spasm of the muscles of both legs and severe rigidity of the right arm were present; the right eye was open, but the left eye was closed; twitching of the right arm and leg was reported.

On September 14 I saw the child in consultation with Dr. M. R. Bennett. The baby appeared ill; he was slightly jaundiced, and was suffering from shock. The left pupil was dilated, while the right pupil was much smaller; the right side of the face was weaker than the left; the anterior fontanelle was bulging; and there was slight spasm of the adductor muscles of the thighs. The circumference of the head was fourteen and a half inches.

A needle was introduced into the subdural space through the anterior fontanelle, and with a serum syringe 30 cubic centimetres of stale blood were removed from the left side; but none was removed from the right side. Magnesium sulphate, in the proportion of 0·2 cubic centimetre of a 25% solution per kilogram of body weight, was given intramuscularly every four hours, and 10 cubic centimetres of the father's blood were injected into each buttock that evening. On September 15 the vomiting ceased and the baby took food better; one cubic centimetre of blood was aspirated from the left subdural space. On September 16 the head circumference was fifteen and a half inches; no blood was aspirated. On September 18 the head was sixteen inches in circumference. The child took the breast and there was much less spasm. No blood was removed by aspiration. On September 20 only slight adductor spasm was present; the eyes appeared normal. On September 21 the baby was weaned and given a diet of "Lactone Syrup". On October 20 the circumference of the head was sixteen inches, and according to the report of Dr. M. R. Bennett the baby now appears normal.

The Hypertonic Infant.

The hypertonic infant is seen frequently, and as individual treatment is so necessary for the satisfactory feeding of infants, I intend to discuss this problem in detail.

During the years of the recent financial depression in Australia I gained the impression that this type of baby was seen more often than usual, and if this is true, then it is probable that the present war with its problems will produce another crop. I do think they are more common in families in which there is a history of neuropathic diathesis in one of the parents.

The baby has a characteristic facies, and its appearance suggests that its age should be months rather than the weeks it really is. There is the tendency to smile readily, and to be very alert mentally; the hands and feet are cold. However, the main characteristic physical sign is the hypertonicity of the muscles, and thus the name hypertonic infant. This baby is subject to abdominal colic (enterospasm), and on this account it cries excessively and is generally unhappy and uncomfortable.

Differential Diagnosis.

The Nervous Infant.—It is difficult to distinguish this baby in all cases; but some assistance may be gained by observation of the attitude of the mother or nurse, who in the case of the nervous infant will undoubtedly overstimulate the baby. The characteristic hypertonicity, cold hands and feet and history of enterospasm will usually determine the true state of affairs. It is wise to make the diagnosis, otherwise the parents may receive some undeserved and unappreciated censure.

Hypothyreoidism.—The cold hands and feet suggest the possibility of hypothyreoidism; but there is not the tendency to constipation, and the characteristic hypertonicity is opposed to the hypotonia seen in hypothyreoidism.

Dyspepsia.—Dyspepsia is a common error of diagnosis. There is a tendency for the physician to switch from one food to another, as he does not realize that enterospasm is a feature of the infant and will probably remain until it reaches the age of five months. The symptoms are suggestive of hyperchlorhydria, and undoubtedly feeding with "Lactone Syrup" milk will make the infant still more uncomfortable. From the gastric analysis in a few cases, however, it has not been possible to confirm the diagnosis of hyperchlorhydria.

Treatment.

The baby should be kept on the breast and should be given one-quarter of a grain of sodium phenobarbital fifteen minutes before feeding, to lessen the spasm. This type of child seems to take the dose in the majority of cases without any ill effects at all, and this drug is preferable in my experience to atropine sulphate. As regards artificial feeding, Czerny-Kleinschmidt butter-flour mixture, in the proportion of three-fifths butter-flour mixture to two-fifths cow's milk, seems to be the ideal food. My experience of giving cow's milk in amounts and calories sufficient to cover the child's requirements fully has not been satisfactory.

Pyloric Stenosis.

As a cause of vomiting, congenital pyloric obstruction is too rarely recognized. I would offer the following suggestions as a safe guide, with the hope that they will be adopted as a means of diagnosis and of saving the lives of many infants, whose condition becomes hopeless before it is recognized, and whose deaths are largely ascribed to other causes.

All cases of vomiting, beginning at birth or shortly thereafter and continuing in spite of a reasonable amount of food manipulation, in breast-fed or artificially fed infants, are to be regarded as cases of pyloric stenosis until this diagnosis can be disproved.

The condition occurs more often in male than in female children (about 80% of reported cases have occurred in males) and as yet there is no satisfactory explanation of this. Grey and Pirie have offered as an explanation the fact that males are generally more vagotonic than females, and that overaction of the vagus may cause a stenosis. This is an interesting hypothesis, but in all probability not the true explanation.

Symptoms appear in three-fourths of the cases during the first three weeks of life. The five cardinal symptoms and physical signs of pyloric stenosis are visible gastric peristalsis, projectile

vomiting, a palpable tumour of the pylorus, constipation and loss of weight.

As regards the question of constipation, I have seen an occasional patient with starvation diarrhœa. Some of the patients who have lost much fat and fluid show a characteristic facies. The forehead, during the waking hours at least, is lined with horizontal furrows, a form of wrinkling associated with pyloric troubles, either stenosis or spasm. The eagerness of the child after vomiting suggests hunger, and is indicated by the open mouth. The sighting of the bottle by the hungry infant instantly produces the sucking reflex, and the mouth shapes itself to receive the nipple.

The tumour is best felt by the examiner sitting down on the left side of the patient and taking up a comfortable relaxed position, so that there is an absence of tension in the tendons of the examiner's fingers. This is essential, as the examiner must be prepared to spend fifteen to twenty minutes if the tumour is to be felt in some cases. Then arrange for the baby to be fed from the breast or bottle, and some minutes later tuck the fingers of the left hand under the edge of the right rectus muscle at a point midway between the costal end of the ninth costal cartilage and the umbilicus, and with the left index finger knead the pylorus. The spasm of the peristaltic waves in the stomach wall then makes the tumour palpable. It generally feels about the size of an olive.

One examination without result does not disprove the diagnosis, and it is advisable to reexamine the baby on similar lines the following day.

Differential Diagnosis.

Pylorospasm.—In pylorospasm the characteristic waves, projectile vomiting and constipation are present, but there is an absence of a palpable pyloric tumour. The diagnosis may be extremely difficult, as in some cases of pyloric stenosis the pylorus is so completely covered by the liver that the tumour is beyond the reach of palpation.

Many of these patients with pylorospasm come to operation; but in a doubtful case it seems to me wise to undertake the operation, if the baby is not making satisfactory progress.

Recently at Mareeba Babies' Hospital, a baby, aged nine weeks, was admitted to one of my beds with a history of pylorospasm. The tumour was not palpable on my first visit, and as the condition of the baby appeared satisfactory, medical treatment was commenced and I decided to examine the baby on my next visit.

My experience in cases of pyloric stenosis at that age had confirmed the views expressed by the late Professor Marriott, of St. Louis, that if a baby with pyloric stenosis reached the age of eight weeks it could be treated successfully on medical lines. However, two days later this baby suddenly collapsed and died, and the *post mortem* examination revealed a definite tumour.

Some of the cases of pylorospasm are associated with hæmatemesis, and under these conditions the vomitus is brown.

The diagnosis of gastric ulcer must then be considered; but according to some German authorities it is possible for hæmatemesis to be present in cases of pylorospasm, when the *post mortem* examination has revealed an absence of ulceration. In my experience these are the most difficult patients to treat, as they do not respond to thick feeding and the use of "Eumydrin" or atropine.

Duodenal Stenosis.—The persistent vomiting of bile-stained fluid by a newly born child should always arouse the suspicion of duodenal obstruction. This vomiting begins upon the first, second or third day of life, so soon as food is taken in considerable quantities. Although vomiting is profuse, it lacks the explosive character of the act in hypertrophic pyloric stenosis. Marked peristaltic waves do not occur. Emaciation and dehydration proceed rapidly, the facial expression becomes anxious, and the infant looks very ill; this contrasts with the eager, alert expression of the baby suffering from pyloric stenosis.

Aerophagy.—All young infants swallow a certain amount of air during and between feedings. Aerophagy may be so excessive as to become pathological and lead to vomiting. Thus severe nasal obstruction, as might be expected in a young infant, interferes with effective suction and causes aerophagy.

A still more frequent source of aerophagy is what may be called the vicious circle of nervous unrest. The reflex act of suction in the young infant proceeds powerfully and effectively only when the child is quiet and at rest. When, on the other hand, the infant is too eager and too excited, as it is apt to be after much hungry crying and persistent sleeplessness, a voluntary effort at suction seems to be attempted and takes the place of the more instructive reflex act. The infant greedily grasps at the nipple, sucking, swallowing, choking and even crying, all at the same moment. Such a procedure rapidly leads to a distension of the stomach with air, and the child ceases its effort to suck and then vomits. One or two grains of chloral hydrate given a few minutes before the meal are usually sufficient to secure a quiet and drowsy state, and are thus effective.

Intracranial Lesions.—Intracranial lesions, such as early hydrocephalus causing projectile vomiting, are frequently missed through neglect to measure the circumference of the skull and to feel the anterior fontanelle.

Treatment.

Reports on the treatment of infantile pyloric stenosis with "Eumydrin" have been published by several observers, all of whom achieved satisfactory results. The technique of this method has not altered to any great extent since the appearance of Svensgaard's paper (*Archives of Disease in Childhood*, Volume X, 1935, page 443). Dr. Elizabeth Svensgaard stressed the necessity of first overcoming dehydration by giving saline solution in the form of subcutaneous injections, consisting of 100 cubic centimetres at a time, once or twice a day. At the same time, and later alone, saline solution is given *per rectum* (75 to 100 cubic centimetres a day

for a couple of weeks). After dehydration has been relieved, "Eumydrin" is given in a 1 in 10,000 solution; five cubic centimetres, corresponding to 0·5 milligramme per dose, are given seven times a day, half an hour before feeding. It is essential to remember that the "Eumydrin" solution does not remain active for longer than ten days. Nourishment was given seven times a day, but only in small quantities (60 to 75 cubic centimetres). This was supplied in the form of breast milk, or milk mixture or buttermilk. When the patient has drunk half the contents of a bottle, a pause in the feed is made and the bottle is removed for five or ten minutes. If vomiting takes place 35 cubic centimetres more are given. Dr. Svensgaard gives the number of days spent in hospital as 69, although Dr. Dobbs (*The Lancet*, January 7, 1939, page 12) in thirteen straightforward cases states that the average length of stay in hospital was fourteen days.

It is necessary to realize that all patients will not respond to "Eumydrin" treatment, and in these circumstances operation is indicated. Combined with this is the fact that to secure good results from this form of medical treatment, it is undeniably of the utmost importance that the nursing should be extremely careful and efficient. When one takes these points into consideration, it is highly suggestive that this form of treatment is only suitable for the odd patient. My experience would bear out this contention, as my results on these lines have been most disappointing.

The following general principles are suggested as a sound working basis for deciding on surgical or medical treatment.

1. On all breast-fed babies, when the test feed reveals a liberal supply of breast milk, perform a Rammstedt operation.

2. If the actual weight of an artificially fed baby is below the original birth weight, then operate.

3. If the artificially fed baby is above its birth weight and nearly eight weeks of age, then give medical treatment a trial. It is necessary, however, to resort to operation if a few days' treatment does not produce definite improvement in weight.

In undertaking medical treatment we must rely on thick feeding, refeeding, the washing out of the stomach once daily with normal saline solution, and the giving of an antispasmodic.

My best results with thick feeding have been obtained when I used lactic acid milk with one drachm of lactic acid to the pint of milk, thickened with 2·5% to 5% of baked flour, and with 5% to 7·5% of added corn cob syrup.

If the food is vomited within one hour the infant is refed with an amount approximately equal to that vomited.

As an antispasmodic give "Eumydrin" atropine sulphate or sodium phenobarbital thirty minutes before feeding.

The results of operations for congenital pyloric stenosis at the Adelaide Children's Hospital have been published by Dr. D. G. McKay in The Medical Journal of Australia of May 27, 1939, at page 813. The details of the cases are as shown in Table I.

Table 1.

Year.	Cases.	Deaths.
1933	7	—
1934	7	1
1935	6	1
1936	13	1[1]
1937	9	1
1938	8	—
Total	50	4

[1] Four months after operation.

Before the Rammstedt operation is performed it is essential to overcome the dehydration by appropriate treatment. In the case of the emaciated infant, however, a blood transfusion is indicated, and the operation should be performed under local anæsthesia. At St. Louis Children's Hospital, where I worked for some months in 1924 with the late Professor Marriott, all the Rammstedt operations were performed under local anæsthesia, and the mortality rate was 1%. If the baby is well nourished the operation can be done under ether anæsthesia with every confidence.

The continuous intravenous drip administration of fluid after operation is not necessary in the majority of the cases, and should be restricted to the extremely wasted baby, who through failure of diagnosis has become athreptic.

Infant Mortality in South Australia.

It is difficult to give a true explanation of the reason of the low infantile mortality figures which have been such a pronounced feature in the South Australian statistics for the last few years. The low figures in my opinion are associated with the high number of calories fed to a large majority of South Australian infants.

W. McKim Marriott in his "Infant Nutrition" (1930) at page 203 states:

> Maintenance of nutrition is one of the important means of preventing diarrhœa. The undernourished infant is especially likely to suffer from diarrhœa and from infections which lead to diarrhœa. Many more infants have died from diarrhœa, which in the ultimate analysis, has been the result of under-feeding, than have died from over-feeding.
>
> Diarrhœa occurring in the case of infants who have been fed properly is in most instances the result of infection somewhere in the body. This frequent type of diarrhœa may be prevented by prompt recognition and suitable treatment of infections as they occur.

The Plunkett system, contrary to physiological principles, has insisted on keeping the percentage of protein of the cow's milk in the region of breast milk protein, and increasing the percentage of fat by the addition of "Kariol Emulsion". This may be suitable in the odd case; but in my experience the baby is inclined to be under-fed, as it is difficult to increase the percentage of fat above 3·2%. For some years the principle of high protein and high sugar percentages, as in "Lactone Syrup" milk, has been adopted by the general practitioner with good

results. This type of feeding is not suitable in all cases, but can be used with advantage in a large proportion. The addition of starch in the form of baked flour to "Lactone Syrup" milk from the onset of artificial feeding should be the practice of the medical profession when this concentrated food is used. The starch undoubtedly helps in the absorption of the protein and salts. When starch gruels are added to cow's milk, the curds precipitated by the action of the acid and rennin are more finely divided than would be the case otherwise.

In some cases in which premature infants weigh three pounds, when breast milk is not available, it is astounding to see the effect of adding small quantities of starch to dilutions of lactic acid milk; the stools are of a much better colour, and the premature infant makes better progress.

At Mareeba Babies' Hospital my practice, when using "Lactone Syrup" milk, is to add baked flour in the proportion of 1·25% in the first two months of life, and in the proportion of 2·5% when the baby is over two months of age.

Diarrhœa in Infancy.

It is impossible with the time available to discuss fully diarrhœa in infancy; but there are some new points of view which should be stressed. Dr. Erwin Schiff, professor of pædiatrics at the University of Berlin, in *Archives of Pediatrics*, Volume LI, at pages 691 and 769, discusses the question of dehydration and intoxication. His experiments led to the remarkable result that anhydræmia by abrupt reduction of water can be obtained only if the food ingested contains protein, while with the same reduction in the intake of water, anhydræmia fails to appear if the food given is free from protein.

Schiff thus arrived at the first conclusion that the decisive factor in dehydration was not only a deficiency in water, but the subsequent disturbance in the intermediary protein metabolism. This logically led him to the second conclusion, that is: for the normal course of the intermediary protein metabolism, the organism requires a definite quantity of water.

The clinical picture, as exhibited in his experimental dehydration, corresponds exactly to the well-known clinical picture of intoxication, while on the other hand all the symptoms of intoxication fail to appear when food free from protein is ingested. Hence his third conclusion: in the genesis of clinical intoxication likewise, the disturbance of the intermediary protein metabolism plays the principal role in the acute deficiency of water in the organism.

In his article Schiff shows that intoxication is due to the abrupt setting in of anoxæmia, which in turn is produced by the acute decrease in the circulating blood. From this it is clear that in order to combat dehydration we should at first eliminate protein from the food and then provide for a repletion of the vascular system in order to counteract the circulatory shock. Clinical experience has shown that diarrhœa may be aggravated and even take a catastrophic course if the sick child should be given a food rich in fat. It is necessary therefore first of all to eliminate fat from the diet.

But, if now protein is also eliminated from the food, the only source of calories left is carbohydrates, if the child is not allowed to fast. From the view-point of fermentation hypothesis, the carbohydrates, of course, should also be left out.

Dr. Erwin Schiff, from his experiments, realized that the fermentation hypothesis was untenable, and years of investigations have assured him that the correct therapy for infants suffering from diarrhœa is to administer carbohydrates.

For several years I have followed the teachings of Dr. Schiff, and I have confirmed his findings. Infants suffering from severe diarrhœa are given his carbohydrate solutions at the beginning of treatment, the same solution of 300 grammes of Ringer's solution being used, mixed with 600 grammes of a 10% rice jelly and 50 to 60 grammes of glucose. At present at Mareeba Babies' Hospital this mixture has been designated "Schiff's solution". One hundred grammes of this solution have a caloric value of about 50. This solution is administered in amounts corresponding to one-sixth of the body weight, but more than 800 grammes per day are never given.

On the day of admission to the hospital the sick infant is given this food only, and if intoxication is diagnosed, the infant is kept on this food exclusively till all the toxic symptoms have disappeared. It is of the greatest importance that milk or milk products should not be given too soon. It is equally important that the first phase of the treatment be carried out irrespective of the condition of the stools.

In addition to alimentary therapy in intoxication, we must replenish the vascular system by venoclysis. The continuous infusion method as employed by Karelitz is recommended. The continuous intravenous drip administration of a 5% solution of glucose in normal saline or Ringer's solution is used. At first 100 to 250 cubic centimetres of this fluid are injected rapidly in 20 to 30 minutes, after which the flow is reduced to a rate of 100 to 200 cubic centimetres, averaging 130 cubic centimetres per kilogram of body weight *per diem*. No child aged twelve months or less receives more than 1,000 cubic centimetres in twenty-four hours. The daily quantity of fluid per kilogram of body weight diminishes as the age of the child increases. At a rough estimate, infants up to one year of age receive the maximum of 1,000 cubic centimetres in twenty-four hours; those aged from one to five years receive 1,000 to 1,800 cubic centimetres, according to the size and age of the children; while older children receive approximately 50 cubic centimetres per kilogram of body weight *per diem*.

The venoclysis is continued until the circulation is improved, diuresis is started and the intoxication

relieved, and until the infant is able to take about two-thirds to three-quarters of its fluid requirement *per os* without ill effects in the gastro-intestinal tract. Dr. Schiff thinks that it is important that the infant should receive liquid *per os*, for dehydration is more efficaciously combated by the oral injection of fluids.

Some authorities stop all fluid by the mouth at the onset of the intoxication; but provided protein is omitted, this is not essential. The stools improve only gradually, as a rule not before the child's state of nutrition has improved; but even if at first the bad condition of the stools should be unaltered, reparation will still take an undisturbed course.

Thus, when the diet to be used for a baby suffering from diarrhœa is under discussion, it is essential in the first place to try to find a cause. In the majority of cases the diarrhœa is due to some parenteral infection, but when the cause is not apparent, and particularly if the infant looks ill or toxæmic, it is essential to follow Dr. Schiff's treatment and omit protein from the diet. The appearance of the baby is the main feature to guide you in your decision.

THE SANATORIUM: QUO VADIS?

By Alan H. Penington, M.D. (Melbourne),
Melbourne.

Since the opening of the first English sanatorium by Dr. George Bodington in the year 1840, there has never been complete unanimity of opinion as to the value of sanatorium treatment for patients suffering from pulmonary tuberculosis. Despite many doubts of the ultimate value of the sanatorium régime in the vast proportion of cases, there have been so many new developments in treatment during the last twenty years that the character of these institutions has greatly changed and is at present in a state of transition. These changes have appeared as the result of new methods of treatment, and a study of the parallel changes in therapy and in sanatorium design and administration since 1840, and of the new methods of treatment lately being followed, will indicate the probable future lines of development of these institutions.

Prior to 1840, patients suffering from pulmonary tuberculosis were incarcerated in over-heated rooms and tended as hot-house flowers. Bodington believed that fresh air and sunlight were beneficial to these patients, and his sanatorium was built on the basis of these principles, only to be ridiculed by both the profession and the laity. He was eventually forced to close the sanatorium owing to lack of support. Nineteen years later Brehmer established the first successful sanatorium in Germany, and many patients regarded as incurable showed a vast improvement after treatment. Within a comparatively short time sanatoria were established throughout England, Europe and America, and the so-called sanatorium routine was elaborated. This routine consisted of rest, fresh air and heliotherapy, with an adequate dietary. The treatment was general rather than specific, being directed to the production of an increased resistance in the patient to tuberculous infection, and concurrently to any other infection. Educational principles, and the segregation of "open" cases from the general population were later principles to be included; the entire function of the sanatorium was confined to these general principles, and no specific forms of therapy were undertaken.

In 1891, Forlanini, in Italy, and Murphy, in Ireland, were able to demonstrate the value of artificial pneumothorax as a method of treatment in pulmonary tuberculosis. The value of this form of therapy was only gradually appreciated, until Saugmann, at Vejlefjord, showed that perfect control of the pulmonary collapse was possible by a combined method of manometry and fluoroscopy. Since 1910, the use of artificial pneumothorax has become progressively more popular; but it has also become apparent that it is merely an ancillary measure in treatment, and must be combined with those methods which produce an increased general resistance to infection.

Sanatoria had therefore to be adequately equipped to carry out the new form of therapy, and the newer institutions required good X ray plants with facilities for fluoroscopy, the requisite instruments and an adequate sterilizing plant.

The increased dependence of physicians on radiography in the diagnosis and progressive observation of pulmonary disease has made it imperative that every sanatorium should have a first-class X ray machine. The subsequent improvement in the understanding of the natural history of pulmonary tuberculosis has led to the revival of some old surgical procedures and to the evolution of newer methods, and for all these differing forms of therapy the modern sanatorium must be specifically equipped. Each patient must receive consideration by a trained staff as to the possible application of these various measures, which range from heliotherapy, through the minor procedures of injections, to the major surgical measure of thoracoplasty. At the same time he must be able to receive the benefit of an organized sanatorium routine, which it is impossible to carry out in the wards of a general hospital. These factors imply the necessity for an adequately equipped modern sanatorium to be staffed with experienced men, and built with operating facilities and wards suited to the nursing of patients subjected to surgical procedures. Only if all these are available in one institution is it possible to fulfil the criteria of continuity of treatment, both in medical personnel and in place, while individual patients with pulmonary tuberculosis are under treatment. At the same time, the administrative costs are considerably lessened if there is no longer any necessity to transfer patients from one hospital to another for the performance of specialized procedures.

Modern medicine has commenced to recognize the significant part played by the psychological factor in the reorientation of a sanatorium patient to the community after the institutional phase of his treatment has been completed. This has led to the widespread introduction of handicraft teaching and of occupational therapy in the sanatorium régime, which plays a large part in the period of rehabilitation. Sanatorium treatment is of economical value only if the patient is returned to the community as an asset and not as a liability; this may be achieved only if a correct estimate of his working capacity is made, and if this capacity is fully employed without detriment to his health. Such an estimate may be made only while he is under medical supervision and his ability and capacity are being evaluated by so-called "grade work" and occupational therapy. These two functions are implicit in a sanatorium, and the institution must be so situated and planned that these functions may be carried out.

A final evaluation of any form of treatment may be reached only as the result of observation over a prolonged period, and it is necessary for every sanatorium to have a department where progressive records of patients' after-histories are maintained. Patients must be taught to return to the sanatorium or to some convenient centre from which reports may be obtained, and periodic analyses of the results of treatment must be made.

The modern sanatorium is no longer comparable to that first planned by Bodington, but assumes the combined characteristics of a sanatorium and a "chest hospital". It is fully equipped to carry out all investigations to establish a diagnosis, it is staffed by physicians who are experienced in radiology and specialized surgical procedures, and it has a first-class operating theatre and X ray department. It is a hospital, but planned for the long-term treatment of each patient on sanatorium principles of rest, fresh air and diet. In addition it educates patients to care for themselves, and assists in the difficult phase of reorientation with the outside world. All these features are designed particularly for tuberculous patients; but they are also those features that are often lacking in the general hospitals which undertake the treatment of non-tuberculous forms of pulmonary disease. The increase in surgical treatment of these latter conditions will soon produce a demand for special hospitals to undertake the long-term care of such patients. Here, in the modern sanatoria, we have such institutions in being, and there is but one obstruction to their use for non-tuberculous people. That obstruction is the wall of prejudice which insists on the institutional segregation of the tuberculous from the non-tuberculous. It can be broken down only if the medical profession as a whole adopts a reasonable attitude to the question of tuberculosis. Let us discard the use of the term "sanatorium", and make our sanatoria model "hospitals for the treatment of pulmonary diseases", thus extending the benefits of the sanatorium to the non-tuberculous instead of confining them to the tuberculous.

Reviews.

THERAPEUTICS.

THE foreword by A. J. Clark, Professor of Materia Medica in the University of Edinburgh, in itself offers an excellent summary of the therapeutic advances that have necessitated the publication of such a book as a "Textbook of Medical Treatment", edited by Dr. D. M. Dunlop, Dr. L. S. P. Davidson and Dr. J. W. McNee.[1] It is composed almost entirely of contributions from members of the Edinburgh School, and is completed by occasional articles from Aberdeen and Glasgow. It need hardly be said that such a volume represents the latest therapeutic authority. It follows traditional lines in opening with a chapter on the general management of a fevered patient and an extensive discussion on symptomatic treatment of pyrexia. The various acute infections are then dealt with, almost in the order of the older text-books, but with the addition of an excellent chapter on chemotherapy.

The sixty pages on tuberculosis, both its prevention and its treatment, constitute one of the best parts of the book. They reveal a balanced view of the various controversial subjects regarding infectivity and pathogenesis and neither too conservative nor unduly radical views on the surgery of tuberculosis.

The section on diseases of the blood includes an excellent chapter on shock associated with blood loss, and here may be quoted one of the many departures from the traditional text-book entry. The dose of morphine in such conditions of shock is stated to be at least a quarter of a grain for an adult and one-sixth to one-twelfth of a grain for children. Such a dogmatic statement will be welcomed by those who for long have thought that the strict application of Young's rule in the treatment of children with shock has simply meant depriving them of the benefits of morphine.

In the chapter on heart diseases we note with some regret the conservative attitude that the Edinburgh School seems to take on quinidine therapy in contrast to the ardent advocacy of this treatment by the London School led by Parkinson.

It is interesting to note in the section on asthma, where an extensive review of the many approaches to the great problem of treatment is given, that the first factor mentioned is the psychological one, next coming that called allergic and then the infective factor and so on. The author of this section agrees with the importance given to the value of respiratory exercises in the treatment of established asthma.

The chapters on rheumatoid arthritis and allied diseases follow closely the reports of recent years of such bodies as the Rheumatism Committee of the British Medical Association. They echo the enthusiasm generally expressed for gold therapy in rheumatoid arthritis by British workers, which contrasts strangely with a large body of opinion in the United States of America.

An excellent section of some thirty pages covers psychotherapy in general practice. It is refreshingly free from psychiatric terminology that is usually inflicted on readers of literature dealing with this subject. Most students and practitioners will also appreciate the concluding chapter in this section, in which details of procedure in treatment are made clear.

A departure from the usual text-book is to be found in the concluding section, which deals with technical procedures and oxygen therapy. Here most of the commoner minor operations are described. With the description of lumbar puncture there is an excellent table summarizing the pathological variations of the cerebro-spinal fluid.

The reader's last impression is doubtless what the authors hoped for: that this is an exceedingly useful and, above all, practical publication.

[1] "Textbook of Medical Treatment", edited by D. M. Dunlop, B.A., M.D., F.R.C.P., L. S. P. Davidson, B.A., M.D., F.R.C.P., M.R.C.P., and J. W. McNee, D.S.O., D.Sc., M.D., F.R.C.P., with a foreword by A. J. Clark, B.A., M.D., D.P.H., F.R.C.P., F.R.S.; 1939. Edinburgh: E. and S. Livingstone. Demy 8vo, pp. 1147. Price: 25s. net.

The Medical Journal of Australia

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Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

TEARS.

To weep is human. We weep in sorrow, from grief, and in distress; we sometimes weep for joy. We weep, some of us, when we come into the world, for has not the cynic said that "the babe is wise that weepeth being born"? Our friends weep when we die. When we are very young we weep from pain, from petulance, from temper, from craftiness or for no clear reason at all. When we are older our tears come not so easily, but they do come; and though they are more likely in our maturity to be due to the woes of another than they were in our youth, they are generally due to self-pity.

But tears have an interest as they are regarded from different angles. Let us take the scientist. The ophthalmologist who wrote learnedly on the hydrogen ion concentration of tears must have been a cold and unimaginative fellow. One may ask what boots it to know the pH value of a tear; yet it might be useful to consider the point. When a lady fair weeps into her 'kerchief, mere man may be undone. It is of little help to him to know that through the ages his fellows have been assailed in this way, that even a sage of such ancient days as Ovid was hardhearted enough to write: "Do not be moved by woman's tears; they have taught their eyes to weep." If he could fix his attention on hydrogen ion concentration he might not yield so easily. The damsel would be out of countenance to find herself pursued by a being with a test tube when she had expected human sympathy and gentle yielding—and the man of science would be the richer. Weeping is one of woman's wiles; she has, however, been warned by Oscar Wilde who wrote that crying is the refuge of a plain woman, but the ruin of a pretty one. In weeping of this kind there is much skill and many a victim is lost in the "sea of melting pearl which some call tears". Attention should be drawn in passing to the peculiar fact that what is weeping in one woman may be snivelling in another. To an Oscar Wilde it may be left to find an aphorism for this. "Nothing dries sooner than a woman's tears." This may be true, but we would not always have it so when we remember that a mathematician wrote of a lass who cried so hard that she soon swam in a sea of her own tears and found "adventures" that ever since have been a joy to children of all ages.

There are tears of such people as those who sang:

> . . . let's pipe our eye,
> Because our Strephon didn't die.

And not far removed are the mythical tears of the crocodile who is said to shed lugubrious tears while he devours his victim in leisure and with relish. It is interesting to note that fanciful though this tale of the crocodile may be, it has enriched medical literature. In 1928 Bogorad described what he called the crocodile tear syndrome, and L. A. Russin has recently reported a case which he calls by the same name and in which a medical student, aged thirty years, suffered from excessive lachrymation every time he ate certain foods.[1] Of course, for those who have no tears "to weep their intermissive miseries" there are always onions.

But these thoughts are discursive and perhaps foolish and they lead nowhere in particular. If we wish to be serious we think of the tears of a heart

[1] *The Journal of the American Medical Association*, December 23, 1939.

bowed down by grief, sorrow or calamity and they are far different. The doctor sees more of these tears than most people, and if he is wise he knows that there is a time to weep as well as a time to make merry—that "to weep is to make less the depth of grief". No one can gauge the suffering of a sorely stricken human soul and it does not help much to be told that tears are tears of self-pity. To know that one has self-pity and to resist may make for self-mastery. Perhaps those who achieve this rare state are content, but the strong and silent and dry-eyed in trouble or sorrow do not seem natural. In any case all tears of this kind are not due to self-pity, and most of us will understand those who can cry with Jeremiah: "Oh that my head were waters and mine eyes a fountain of tears." Most of us also, whether we treat patients or not, will sometimes urge an opening of the flood gates of the emotions with counsel to "have a good cry", knowing that calm will follow. This calm will be akin to that of Henri Frederic Amiel's friend, who wept on his shoulder—"her heart was unbosomed of its grief, disburdened of its weight, almost like that of a penitent absolved by her confessor". But some hurt is much too deep to be helped in this way—the broken heart is not easily recovered, nor the crushed spirit revived. And such suffering is widespread. In fact not since the dark ages have there been so much bitter pain and anguish as are abroad in the world today. Cruelty, stark, deliberate and terrible, is claiming thousands of victims; and their tears avail them not! Almost are we compelled to adopt the pessimism of Douglas Reed who thinks that he shall "see weeping in these years to come, more and more like a . . . flood and no hope of stopping it . . . but only more and more tears". In Australia we are remote from these happenings and are in danger of allowing them to pass us by unmoved. Even though we may perhaps be able to do little or nothing to ease the lot of these stricken people in other parts of the world, we can, if we will, cultivate the gift of charity—not the fashionable pseudo-virtue that passes by that name, but true charity, the love of man for his fellows, the virtue which, above all others, sweetens and ennobles human life.

Current Comment.

CONGENITAL LEUCHÆMIA.

In spite of recent advances in the study of the blood and blood-forming organs, the leuchæmias remain a baffling and mysterious group of diseases. Any observation that may throw light on their nature and origin is of great importance. The occurrence of congenital leuchæmia is of peculiar interest, for here the disease may be studied in miniature and from the beginning. Weston M. Kelsey and Dorothy H. Andersen summarize the literature and present the history of a case of congenital leuchæmia.[1] Their patient, a boy, was admitted to hospital when thirty days old, with a history of purpura and fever of seven days' duration. The parents were healthy; there was no family history of a tendency to bleeding, and the child appeared normal at birth, except for a forceps mark over the right temporal region. He remained well until the onset of purpura twenty-three days after birth. On admission to hospital he appeared desperately ill. Massive ecchymoses and hæmorrhagic bullæ covered the whole body. The superficial lymph glands were enlarged, and the abdomen was practically filled by the liver and spleen, the former reaching almost to the *symphysis pubis* and the latter to the iliac crest. Examination of the blood revealed 6·5 grammes of hæmoglobin per hundred cubic centimetres, 2,000,000 red blood cells and 225,300 white blood cells per cubic millimetre, 2% polymorphonuclear cells, 22% myeloblasts, 68% myelocytes and 2% lymphocytes. Platelets numbered 110,000 per cubic millimetre and the bleeding time was prolonged. The child died two days after admission to hospital. The authors give a detailed account of the autopsy findings. The most striking finding was the presence of an infiltration of large cells throughout the various tissues. These cells had large, pale, finely granular nuclei, and a scanty cytoplasm with a distinct cell membrane; they were found in the blood and they infiltrated most of the organs. A very complete histological examination was made, but the authors' technique was apparently not sufficiently good for these peculiar cells to be identified with certainty. It is indeed a difficult matter to fix and stain tissues of this nature. Methods which are adequate for ordinary histological examination do not protect immature blood cells from shrinkage. The authors discuss the differential diagnosis, laying stress on the distinction between congenital leuchæmia and *erythroblastosis fœtalis*. But until better histological techniques are available, this distinction will not be easy, at any rate in some cases.

Nine instances of congenital leuchæmia have been established; in the authors' opinion two at least of these may have been cases of *erythroblastosis fœtalis*. Possibly there is some link between the two groups of dyscrasias. Their occurrence in the newly born

[1] *American Journal of Diseases of Children*, December, 1939.

suggests the importance of hereditary or familial influences, or possibly of antenatal nutrition. There are, of course, some competent observers who attribute both conditions to the presence of infection, but it has not been possible to establish this view.

EXOPHTHALMOS IN GRAVES'S DISEASE.

An interesting case history is reported by W. Russell Brain in *The Lancet* of December 9, 1939. The patient under discussion had had syringomyelia for thirteen years before she developed thyreotoxicosis associated with her first pregnancy. This was cured eventually by medical treatment. The importance of the case lies in the fact that a person with bilateral cervical sympathetic paralysis developed exophthalmos. This paralysis was more pronounced on the right side than on the left, but, as is commonly found in thyreotoxicosis, the exophthalmos was more pronounced on the left side.

Our ideas concerning exophthalmos in thyreotoxicosis have undergone changes during the last few years. Not so long ago it was believed that the cause was to be found in increased retrobulbar fat, though why fat should increase here and diminish everywhere else remained a mystery. Those who waxed enthusiastic over the contraction of Müller's muscle did not take great pains to describe either its anatomy in man or its mechanical capacity for causing exophthalmos. It has been known for some time that repeated stimulation of the cervical sympathetic trunk (for example, in a cat) can cause the symptom complex of Graves's disease, including exophthalmos. Following this the opinion was held for some time that the thyreoid activity was due to stimulation of its sympathetic nerve supply and that the exophthalmos was also due to sympathetic influence on the eye muscles. We are tolerably certain now that the former conclusion is incorrect, that the thyreoid hyperactivity in this experiment is due not to nervous influence on this gland, but on the pituitary gland, which is caused to produce excess thyreotropic hormone. No experiment or operation has yet been successful in mitigating thyreotoxicosis by severance of cervical sympathetic nerves. It is also believed that the eye signs are not due to cervical sympathetic nerve activity. It is true that, by this means, widening of the palpebral fissure can be produced. Neither physiologists nor physicians have been able to explain why, if this is the mechanism in the human subject, there is no dilatation of the pupil in the typical exophthalmic patient. Nor has anyone been able to influence the exophthalmos, in man or laboratory animals, by cutting the sympathetic nerve supply to the eye.

The case described by Brain is a peculiar but important substantiation of these opinions. Here is a patient with cervical sympathetic paralysis who subsequently develops exophthalmos. Many experiments have been carried out in the last two or three years, establishing the connexion between exophthalmos and pituitary activity. For example, G. G. Smelser[1] has shown that injection of pituitary extracts causes protrusion of the eye because of an extensive œdema of the orbital connective tissue. This effect is enhanced if the thyreoid gland is first removed. The lower the basal metabolic rate, the better the result. We know that no obvious connexion exists between the degree of exophthalmos and the severity of the thyreotoxicosis.

ADRENAL GRAFTING IN ADDISON'S DISEASE.

Gland grafting in endocrine hypofunction is an attractive form of treatment; but, unhappily, it is rarely successful. Glandular tissue, placed in an abnormal situation and provided with an inadequate blood supply, tends to atrophy. It does not often retain its function. The successful grafting of adrenal tissue has been performed so seldom that the report of a case seems to be worthy of comment. Hamilton Bailey and Kenneth D. Keele have recently recorded the cure by adrenal grafting of a patient with Addison's disease.[2] The patient, a woman, was first shown before the Royal Society of Medicine in November, 1935, some five months after the grafting of fœtal adrenal glands into the sheaths of the *recti abdominis* muscles. The progress was satisfactory throughout, and the woman was able to work long hours as a cook. Her weight remained steady. The systolic blood pressure was constant at about 130 millimetres of mercury and the diastolic at 80. The menopause occurred in April, 1938, when the patient was forty-eight years old. In February, 1938, the sodium content of the serum was estimated at 342 milligrammes per 100 cubic centimetres, and the potassium content of the serum at 19·95 milligrammes per 100 cubic centimetres. Pigmentation decreased slightly during the four years after operation, but was still apparent in April, 1939. Bailey and Keele state that criticism of their report has been of two main types. First, doubt has been expressed concerning the diagnosis, and, secondly, it has been suggested that spontaneous cure occurred. In reply to these arguments Bailey and Keele state, first, that the diagnosis was accepted by the Clinical Section of the Royal Society of Medicine in 1935, and, secondly, that "it is unlikely that a spontaneous cure would occur so closely to the event of grafting". Presumably they mean that the occurrence of spontaneous cure so soon after grafting would be such a remarkable chance as to be scarcely worth considering in an assessment of probabilities. On the evidence presented it would seem that a cure has been effected in this case. But one can readily conceive of other possibilities. It is to be hoped that the patient will be kept under observation until her death and that a *post mortem* examination will be made.

[1] *American Journal of Pathology*, May, 1939.
[2] *Proceedings of the Royal Society of Medicine*, September, 1939.

Abstracts from Current Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

The Value of the Complement Fixation Test in the Diagnosis of Psittacosis.

K. F. MEYER AND B. EDDIE (*The Journal of Infectious Diseases*, November-December, 1939) undertook experiments with antigens prepared from cultures of the virus of psittacosis in Rivers-Li medium and mouse spleen antigens for use in the complement fixation test in the diagnosis of psittacosis. Details of the preparation of both these antigens are given. It was found that by the use of culture antigens serum of animals hyperimmunized with mouse tissues could be titrated without the occurrence of fixation reactions attributable to antibodies produced by mouse proteins, and that mouse spleen antigens could be used in the estimation of antibodies in serum of animals hyperimmunized with cultures. It was proved that only organs rich in virus elements produce antigenically potent mouse spleen antigens. Serum in dilutions less than one in four from infections proved bacteriologically to be due to psittacosis invariably gave positive reactions for many months after convalescence of the patient, and the antibodies were found to persist in some cases for over one and a half years. It was also found that a psittacosis infection could be diagnosed with considerable certainty as early as the sixth to eleventh day, at a time when clinical signs are indefinite, sputum scanty or unobtainable and epidemiology not proved. But since the specific complement fixation may not be definite even at the end of the fourth week, serum from a patient suspected of being infected with psittacosis virus, if giving a positive reaction in dilutions of one in two and one in four, should be retested within five to ten days. The specificity of the tests is under investigation, since weak reactions were sometimes obtained with the serum of patients suffering from febrile conditions, such as typhoid fever or undulant fever. It had also been noted that serum yielding reactions with the Wassermann test occasionally gave reactions. The majority of sera obtained from normal healthy persons rarely gave an appreciable reaction. It is suggested from observations made that convalescent serum may shorten the course of the disease and that highly potent goat serum may be equally effective.

A Virus-Inactivating Agent from Human Nasal Secretion.

F. M. BURNET, DORA LUSH AND A. V. JACKSON (*The British Journal of Experimental Pathology*, October, 1939) report experiments "dealing with the power of serous nasal secretion from normal human beings to inactivate influenza and certain other viruses". The nasal secretion was obtained by pushing a roll of gauze or gauze and cotton-wool into each nostril, in some cases after spraying with 50% glycerol-saline solution to promote secretion. When uncomfortably wet, the plugs were removed and wrung out over an open Petri plate. Usually about one cubic centimetre of clear non-viscous fluid was obtained. This was centrifugalized and rendered sterile by heating for thirty minutes at 56° C., or by filtration through gradocol membrane. In testing for virus-inactivating power, equal parts of nasal exudate and virus, suitably diluted in saline solution containing 10% normal horse serum, are mixed and incubated at 36° C. for some hours. Control mixtures of dilute virus and saline solution are exposed to the virus content on the chorio-allantoic membrane or in the appropriate laboratory animal. It was found that whereas the interaction between virus and antibody is a rapid one, the action between virus-inactivating agent and virus proceeds more slowly, and at refrigeration temperature shows little change in five hours. The virus-inactivating agent was proved capable of inactivating the viruses of influenza, louping-ill, herpes, B. virus and probably Rous sarcoma virus. It has no action on viruses of vaccinia, ectromelia, fowl-pox, infectious laryngo-tracheitis, myxomatosis, psittacosis or pseudorabies. The action on the virus of poliomyelitis is slight but significant. The virus-inactivating agent is rendered almost completely inactive by boiling for ten minutes.

The Characters and Systematic Position of Morgan's Bacillus.

A. SEVIN AND R. BUTTIAUX (*The Journal of Pathology and Bacteriology*, November, 1939) undertook an investigation to determine whether Morgan's bacillus number 1 is possessed of distinctive differential characters. The five strains studied were obtained from: (i) ulcerated lesions of the rectum in a female aged thirty-five years, (ii) urine in a case of hæmaturia occurring in a male aged fourteen years, (iii) duodenum in a case of cholecystitis and colitis in a male aged thirty years, (iv) a case of cholecystitis in a male aged fifty years, and (v) mesenteric abscess in a female aged sixty years. Three of the strains were recovered in pure culture. The authors formed the following conclusions: (1) Morgan's bacillus number 1 differs from the other types described in possessing distinctive characters which are fairly constant in different strains and are not altered by long maintenance in artificial media. (2) It fails to ferment lactose and mannitol, and ferments glucose, fructose and usually galactose, with very slight gas production. (3) Cultures on 1% agar develop as a film on the surface, but not on 3% agar. This character is also seen in certain O forms of Proteus, but not in other intestinal organisms. (4) Strains of Morgan's bacillus show a community of antigens, most marked in cultures on soft agar; such cultures show also serological relationships with certain strains of *Proteus vulgaris* and Proteus X19. (5) It is considered that Morgan's bacillus number 1 is to be regarded as a distinct organism related to *Bacillus proteus* and included in the subspecies which does not ferment mannitol.

Routine Laboratory Examinations for Corynebacterium Diphtheriæ.

C. ALFRED PERRY AND ELIZABETH PETRAN (*The Journal of Laboratory and Clinical Medicine*, October, 1939) emphasize certain points in the successful routine examination for *Corynebacterium diphtheriæ*. In the choice of media, Löffler's and serum tellurite agar are compared, and preference is given to the latter, as on it colonies of *Corynebacterium diphtheriæ* can usually be identified by simple inspection of inoculated plates. Since, however, a considerable percentage of cultures occur on Löffler's medium and not on tellurite plates, it is the authors' custom to employ both. The authors lay stress on the importance of careful preparation of Löffler's medium, with particular attention to pH, time, pressure and air conditions during autoclaving. The stain employed is Löffler's methylene blue. Direct smears of material obtained on swabs have proved satisfactory, but experience in this field is required and cultures should also always be attempted. The high incidence of nasal diphtheria (nearly one-third of the cases in the authors' series) is emphasized.

Active Immunization against Undulant Fever.

JOHN A. KOLMER AND AMEDEO BONDI (*Journal of Immunology*, November, 1939) have studied the problem of active immunization against undulant fever. Rabbits were immunized with a heat-killed suspension of *Brucella abortus* and the serum obtained was tested for agglutinin and opsonin content and for protection value in mice infected intraabdominally with lethal doses of virulent *Brucella abortus* and *Brucella melitensis*. Other rabbits were immunized with a heat-killed vaccine of *Bacillus typhosus*, and others again with vaccine of *Brucella abortus* and *Bacillus typhosus* simultaneously. The results obtained indicated that rabbits produced agglutinins for *Bacillus typhosus* more effectively than for *Brucella abortus*, and that rabbits immunized with vaccines of heat-killed *Bacillus typhosus* and *Brucella abortus* simultaneously produced agglutinins for both organisms in the same degree as rabbits immunized with the single vaccines. The authors discuss the possibility of effective simultaneous

immunization of human beings against typhoid and undulant fevers with a mixed heat-killed vaccine, and regard the indications as encouraging.

HYGIENE.

Tuberculosis Case Finding in a Large City.

BRUCE H. DOUGLAS AND GAIUS E. HARMON (*American Journal of Public Health*, June, 1939) state that in Detroit the health department arranged for the examination by private medical practitioners of every person in a quarter of the city in which tuberculosis was common, the city paying for the examination of those who could not afford to pay. Of 48,472 tested with tuberculin, 27·1% gave positive reactions; and of those with positive reactions, 18·1% were shown by X ray examination *et cetera* to be suffering from active infections. In other districts, where only contacts and suspects were tested, higher figures, 33·9% positive, were obtained in the tuberculin test, and 24% proved to be active sufferers. Thus mass testing suggests that about two in every 1,000 of the city population may be active sufferers from tuberculosis. Altogether 114,800 persons were tested by 705 medical practitioners. Contacts gave 41% positive results to tuberculin tests, suspects gave 32·5%, and others 26·5%. The proportion of positive results rose with age to 50% at fifty years from 6% among those under ten.

Deaths in Ontario Attributed to Diabetes.

R. D. DEFRIES AND A. HARDISTY SELLERS (*Canadian Public Health Journal*, September, 1939) state that in Ontario insulin has been made available by the health department when the medical attendant certifies that the patient is unable to pay for it. Defries and his co-workers report on the deaths from diabetes in 1935 and on the information provided by 88% of the medical practitioners furnishing the death certificates. Of the 456 persons officially recorded in 1935 as dying from diabetes, 70% used insulin at some time and 24% used none. In 1930, 56% had used insulin. Of those who used insulin, 38% used it regularly, 15% had discontinued its use, 20% used it irregularly; 27% used it only at the point of death. Of the group as a whole, 27% used insulin regularly. Most of those who did not use insulin belonged to the group over seventy years of age (13%). Of the 456, only 60% were under constant medical supervision, and less than one-half cooperated with the medical attendant. Of the 602 persons followed up and in regard to whom diabetes was mentioned on the death certificate, 146 died from other causes, including *angina pectoris* 53, cancer 27, tuberculosis 17. Of the 456 officially allotted to diabetes, only 291 would have been so allotted had the opinion of the medical attendant been ascertained. The true picture of diabetes mortality and its trend cannot be secured until medical practitioners definitely indicate on their certificates its relative importance as a cause of death.

The Need for a Campaign against Ragweed.

H. B. ANDERSON (*Canadian Public Health Journal*, September, 1939) writes that of the people in North America 3% to 5% are hypersensitive to wind-borne pollen. The figures for hay fever in late summer and early autumn are increasing, and more than 80% of the sufferers are sensitive to ragweed. The enormous increase of hay fever in autumn all over America coincides with the spread of ragweed. It forms a great belt, originally east of the Mississippi and in the northern States, but now has extended westward and southward. It has invaded Canada and threatens many summer resorts in Ontario. Deforestation, floods, soil erosion, insects, transport wagons, all have helped this noxious weed to spread. Of hay fever patients, 30% suffer from respiratory complaints and may be droplet carriers, while the sneezing helps to spread infection. An educational campaign for the elimination of ragweed is advocated.

The Pathogenesis of Infectious Myxomatosis (Sanarelli) as Modified by Certain Immunizing Agents.

ROSCOE R. HYDE (*The American Journal of Hygiene*, July, 1939) writes on the pathogenesis of infectious myxomatosis as modified by certain immunizing agents. During ten years not a single rabbit of 2,000 survived this virus infection, death occurring in eight to eleven days. Intradermal injection of heat-inactivated tissue virus (60° for 30 minutes) gives some degree of protection. Five out of ten survived, and in three others death was delayed. The immune response appears to be enhanced by the addition of the viable type III pneumococcus or *Bacterium lepisepticum*, but not by vaccinia or fresh rat tumour tissue. Young rabbits respond to the treatment better than old.

Effect of Alcohol on the Kidney.

MAURICE BRUGER *et alii* (*The Journal of the American Medical Association*, May 6, 1939) give the results of the experimental administration of alcohol to 21 patients, 16 of whom were suffering from renal disease. These studies do not answer the question as to the part played by alcohol in the causation of Bright's disease. It is significant that acute and chronic diffuse glomerular nephritis is not aggravated by alcohol and whisky in the doses given, namely, 50 to 200 cubic centimetres of a 25% solution by volume of pure ethyl alcohol. A transient deleterious effect is observed frequently in arteriosclerotic nephritis. It is pointed out that there is a peripheral vasodilatation after the administration of alcohol, and there may be a redistribution of blood, which will diminish the flow of blood through the kidney. In arteriosclerotic nephritis, diverting blood from the kidney to the skin may aggravate the renal lesion, since in such cases the flow of blood is already impaired by vascular sclerosis. With glomerular lesions, on the other hand, particularly the acute diffuse glomerular nephritis, such a redistribution of blood would tend to reduce congestion in the glomeruli, and the taking of alcohol in moderate doses may actually be beneficial. The present studies show that alcohol increases the output of urine, even in the presence of decreased functional capacity of the kidneys. Alcohol has no deleterious effect on the kidneys of normal subjects.

Blood-Bank Bank Books.

THE Scottish correspondent of *The Lancet* (*The Lancet*, October 7, 1939) writes that the organizers of the Scottish national blood transfusion service, partly in order to stimulate the enrolment of further donors, have introduced a system of "bank books". Depositors of blood are given books in which each donation of blood is recorded. The blood given is labelled with the name and number of the donor, and as soon as it is used a certificate giving particulars of the person to whom it was administered is sent to the donor for pasting into his or her bank book.

Silicosis in a Dental Technician.

L. E. SILTZBACH (*The Journal of the American Medical Association*, September 16, 1939) reports the history of a dental technician whose only occupation for nineteen years had consisted of polishing dentures with an abrasive powder called "pummy", and who was found to have advanced silicosis.

Physical Exertion and Coronary Occlusion.

A. M. MASTER, SIMON DACK AND HARRY L. JAFFE (*The American Heart Journal*, October, 1939) present the results of a study of the activities associated with the onset of acute coronary artery occlusion in 1,077 patients who had experienced 1,440 attacks. The cases were classified in regard to occupation, the activities existent at the time of occlusion, the time of day at onset, and the relation of associated factors to the onset, such as meals, excitement, surgical procedures and infection. The circumstances preceding the onset of symptoms included sleep, rest, mild activity, moderate activity, walking, unusual exertion, meals, emotional excitement, surgical operation and infection. None of these factors, with the possible exception of surgical procedures, played any role in the pathogenesis. There is no evidence that physical effort or excitement produces intimal hæmorrhage in the coronary arteries, which is the usual antecedent of thrombosis and occlusion.

British Medical Association News.

SCIENTIFIC.

A MEETING of the South Australian Branch of the British Medical Association was held on November 30, 1939, at the Institute of Medical and Veterinary Science, Adelaide, DR. M. ERICHSEN, the President, in the chair.

Some Problems of the First Year of Life and their Management.

DR. F. N. LE MESSURIER read a paper entitled "Some Problems of the First Year of Life and their Management" (see page 336).

DR. M. T. COCKBURN thanked Dr. Le Messurier for his interesting paper and congratulated him on the manner of its presentation. He said it was the more valuable because it was based on Dr. Le Messurier's own experiences in practice. In dealing with hæmorrhagic disease of the new-born and the decision between giving blood by the intramuscular route or by transfusion, Dr. Cockburn said that the more he saw of this disease, the more he felt that blood transfusion should be given in every case at the earliest possible moment. He could not remember one death having occurred during the last few years in cases in which this had been done. He also stressed the importance of the continuance of breast feeding in these cases.

Dr. Cockburn said he was interested in the reports of the two cases of subdural hæmatoma, and congratulated Dr. Le Messurier on the very satisfactory results obtained in these cases. The dosage of sodium phenobarbital quoted in the treatment of the hypertonic infant had often been queried by the general practitioner as being too large; but Dr. Cockburn's experience was in line with that of Dr. Le Messurier, and he believed that unless one large dose were given little therapeutic effect could be expected. He had given a baby, aged six months, with hydrocephalus, three and a half grains of "Luminal" a day for two months without detrimental effect; the baby lay quietly, but roused and took its bottle readily.

Dr. Cockburn thought that the butter-flour preparation, to be successful, seemed to require skilled preparation for the best results to be obtained, and although Dr. Cockburn often employed butter-flour in the dietary management of the hypertonic infant, it was by no means a simple matter. While Dr. Cockburn was greatly interested in Dr. Svengaard's reports on "Eumydrine" in the treatment of pyloric stenosis, his experience in its use coincided with that of Dr. Le Messurier. Dr. Cockburn considered that it was of little, if any, avail in true cases of congenital pyloric stenosis. Since he had been using the solution described by Professor Schiff in the treatment of toxic diarrhœal conditions, had excluded fat and protein from the dietary, and in some cases had employed the intravenous infusion of fluids by slow drip, more cases had reached a successful termination than before this type of treatment was employed. The treatment of these patients, in Dr. Cockburn's opinion, was one of the most difficult problems the pædiatrician had to face. Whilst they had reason to be proud of the low figure shown in the infant mortality statistics in South Australia, an analysis of those figures showed no decrease in the number of deaths on the first day and in the first week of life. Dr. Cockburn drew members' attention to the ever-present problem of the premature infant; he asked for the exercise of every care before, during and immediately after the birth of such an infant.

DR. L. LLEWELLYN DAVEY thanked Dr. Le Messurier for his interesting paper. He mentioned the case of a young infant that he had attended; free hæmorrhage from the umbilicus had occurred after separation of the cord. The bleeding had ultimately been controlled by completely closing in the umbilical depression with five or six Michel's clips. The same baby was being fed from a bottle with a teat but no valve. Dr. Davey considered that that was a bad method, as the infant had to suck very hard and had frequently to release the teat to allow air to enter the bottle. This baby vomited blood on several occasions, mixed with its drinks, and on examination of its mouth and soft palate petechial hæmorrhages could be seen. It was therefore obvious that the child was sucking blood out of its own mucous membranes, swallowing it and then vomiting it. When it was fed from a bottle with a valve at the other end, all hæmatemesis ceased.

A MEETING of the Victorian Branch of the British Medical Association was held on September 20, 1939, at Saint Vincent's Hospital, Melbourne. The meeting took the form of a series of clinical demonstrations by members of the honorary medical staff of the hospital.

Splenic Anæmia.

DR. W. J. NEWING showed a female patient, aged eighteen years, suffering from splenic anæmia with unusual features. She had come under notice twelve months earlier because of severe and prolonged menorrhagia, which had brought about a reduction in the number of erythrocytes to 2,000,000 per cubic millimetre. Investigation along gynæcological lines had failed to reveal the cause of the hæmorrhage. On two occasions one pint of blood had been given by transfusion and radium had been applied. The bleeding ceased and no further menstruation occurred for four months. It was observed at the outset that the spleen was enlarged, extending a hand's breadth below the costal margin; the liver was also enlarged downwards by a finger's breadth. No enlargement of lymphatic glands was found. The blood serum failed to yield the Wassermann reaction. The leucocytes numbered 4,000 and the platelets 200,000 per cubic millimetre. The appearances in the blood films were not normal; the corpuscular fragility was not affected; and there were no features in the family history suggestive of a tendency to bleeding.

After prolonged rest and vigorous antianæmic therapy the erythrocytes had risen in number only to 3,200,000 per cubic millimetre, and the hæmoglobin value had not become higher than 55%. In April, 1939, ascites had developed, with the characteristics of a transudate, and it had disappeared spontaneously; attempts to grow tubercle bacilli from the fluid had failed. From January, 1939, the patient had had heavy menstrual losses of blood; but the loss had diminished gradually and latterly the periods had become regular and normal in amount. The spleen had receded somewhat and the liver was just palpable; despite intensive treatment the reticulocytes had not exceeded 5% and the differential counts of the leucocytes had been consistently within normal limits.

Dr. Newing said that severe anæmia was uncommon at so early an age, and that menorrhagia was not usual in splenic anæmia. In spite of the report on the blood serum he thought that syphilis might be an ætiological factor and appropriate therapy was to be undertaken. His experience of splenectomy in splenic anæmia had been disappointing, and after surgical consultation, splenectomy had been deemed inadvisable.

Bronchogenic Carcinoma.

Dr. Newing also showed a male patient, aged seventeen years, suffering from collapse of the upper lobe of the right lung. The boy had had an illness of two months' duration resembling a severe cold, with copious expectoration, loss of weight and heavy sweating. Those symptoms had abated greatly and the course had become afebrile. Repeated examination of the sputum for tubercle bacilli had been carried out with negative findings; a typical picture of collapse of the upper lobe was demonstrable from the skiagrams; the lower edge was sharply defined at the interlobar fissure. The physical signs were typical of cavitation in the affected area, but no lymphatic glands were palpable.

Dr. Newing said that, although at first sight the lesion appeared to be tuberculous, lobar collapse was not common

in that disease and tubercle bacilli had not been recovered from the patient. Though lipiodol had not been introduced, as the patient had only been in hospital for one week, he thought it more than likely that the collapse was due to the presence of bronchogenic carcinoma.

Hypochromic Anæmia.

DR. C. H. FITTS showed two patients, each suffering from severe hypochromic anæmia due to rare and somewhat obscure causes. Although the anæmia was obvious, the symptoms complained of were not relevant to this sign. The first patient was a man, aged seventy-three years, an old-age pensioner, whose mental and physical alertness contrasted strikingly with his extreme pallor.

On July 12, 1938, he had complained of discomfort in the epigastrium following a meal, particularly if he walked about immediately afterwards. He also complained of flatulence and of his "nerves". Almost as an afterthought he mentioned that for many years he had had recurrent epistaxis, and that ten years previously he had had the veins removed from his nose. Despite this, and the taking of tablets to make the blood clot, his nose still bled at times. He would not admit that the hæmorrhages had ever been severe.

On physical examination at that time his pallor was extreme. Against this background telangiectases were obvious, widely scattered over the skin of the face and the mucous membrane of the lips. His finger nails were concave to a degree that Dr. Fitts had never seen approached. There were no other abnormal physical signs, though some weight had been lost.

A diagnosis of hereditary multiple telangiectasia was made, a rare disease first described by Rendu, and later, and more fully, by Osler in 1901. The familial nature of the disease in this patient, though not the hereditary nature, was established by a report from Ballarat of a first cousin who had recently died from recurrent epistaxis and anæmia. The hereditary factor was supported by the patient's picturesque description of two uncles with white beards, which he often remembered stained red with blood.

Dr. Fitts said that it was worthy of record that this man's complaint was of indigestion, and the second patient recorded by Osler (the brother of his first patient) died from a carcinoma of the stomach. He had had recurrent epistaxis, and at autopsy multiple angiomata had been found in the stomach.

Dr. Fitts's patient had a hæmoglobin value of only 33%, a red cell count of 3,000,000 and a white cell count of 5,000 per cubic millimetre, and pronounced hypochromia of the red cells. In hospital he made excellent progress, and his blood approximated to normal on his discharge. He had now returned to hospital, and as a result of the insidious loss of blood from the nose, his hæmoglobin value was 45%, and his red cell count was 4,200,000 per cubic millimetre.

As recently as 1932 there were less than 600 recorded cases in 100 families in the literature. Dr. Fitts said that there were three families known to him in Melbourne, and it was probable that the condition of many patients went unrecognized. Epistaxis and bleeding from the mouth and gums were commonest, but there was no site of external hæmorrhage from which bleeding had not been reported and where the telangiectases had not been found. It was remarkable that so many of the patients were first recorded in and beyond the sixth decade.

Dr. Fitts's second patient was a young woman, aged twenty-eight years, a semi-trained nurse. Her complaint was of lassitude and irritability. She had had amenorrhœa for seven years, and had not really felt well during that period. Twelve months previously, while she was having her eyes tested, the oculist had told her that she was anæmic. She then consulted her doctor, and was found to have a hæmoglobin value of 27%. She was treated, according to her statement, as suffering from idiopathic hypochromic anæmia. A course of Blaud's pills restored her hæmoglobin to a level of 78% and she felt better.

On examination she was grossly anæmic. Physical examination revealed no abnormality, except for the pressure of a firm mass which was immovable in the right iliac fossa. This mass was slightly tender, and the patient then recalled slight attacks of pain in this region, of no very definite character. Pelvic examination revealed no abnormality. There were no bowel symptoms, but the result of the occult blood test was strongly positive in the fæces.

Examination after a barium meal and barium clysma revealed the presence of a persistent filling defect in the first part of the ascending colon. The blood examination revealed an anæmia of the hypochromic type with a hæmoglobin value of 33%. There was no response to the Mantoux test (1/1,000). X ray examination of the chest revealed no abnormality, and there were no deposits in the bones. The patient's weight had fallen over four years from thirteen stone to eight stone eight pounds.

The diagnosis appeared to lie on clinical grounds between carcinoma of the ascending colon, ileo-caecal tuberculosis, and perhaps regional ileitis. The evidence seemed to be most in favour of carcinoma of the ascending colon. As soon as the patient's condition warranted it was proposed to perform a laparotomy.

Aortic Stenosis and Regurgitation.

DR. T. A. F. HEALE showed a man, aged twenty-four and a half years, who was known to have had an abnormal heart since 1925, when, at the age of ten years, he had been referred by the school medical officer to the Children's Hospital. He had had no symptoms then and was able to play strenuous games such as football without distress. There was no history of rheumatic infection; physical examination had revealed no abnormality except for the heart. The heart findings were the following: (i) the apex beat was in the sixth intercostal space, one and a half inches outside the nipple line; (ii) a systolic thrill could be felt with maximum intensity at the aortic area; and (iii) a systolic murmur and a diastolic murmur could be heard all over the precordium, with maximum intensity at the aortic area. A tentative diagnosis of congenital heart disease was made.

In 1934 the patient had attended at another hospital, and the same signs had been found on examination. An X ray examination of the heart had revealed an enlarged left ventricle and a rounded bulge anteriorly and to the right in the region of the ascending aorta. A diagnosis of congenital aortic stenosis was made.

Dr. Heale said that the patient had attended at Saint Vincent's Hospital in 1938, complaining that he became slightly breathless on exertion, though he was still able to play a moderately strenuous game of tennis for twenty minutes without distress. He was a tall, slightly built young man of moderately good physical development. From examination it was found that the heart signs were the following: (i) The apex beat was in the sixth intercostal space, one inch outside the nipple line and four and three-quarter inches from the mid-sternal line. (ii) Bulging of the chest wall over the precordium was noted. (iii) A systolic pulsation in the second right intercostal space near the sternum was visible. (iv) A rough systolic thrill in the aortic area could be easily felt. (v) A loud, rasping, systolic murmur and a diastolic murmur were heard all over the precordium, with maximum intensity in the aortic area. (vi) The systolic blood pressure was found to be 160 millimetres and the diastolic 60 millimetres of mercury. (vii) On X ray examination enlargement of the left ventricle and aneurysmal dilatation of the ascending aorta were considered to be present. There was no radiological evidence of enlargement of the left auricle, nor was distortion or obstruction of the œsophagus seen when barium was being swallowed. (viii) In the electrocardiograms no significant changes were observable. The blood serum did not react to the Wassermann test.

Dr. Heale, in summarizing the case, stated that the patient had considerable enlargement of the heart, aortic stenosis and aortic regurgitation and aneurysmal dilatation of the ascending aorta. He said that the chief interest

was in the ætiology of those structural changes. Congenital heart disease was suggested by the long history with almost complete absence of symptoms, and by the physical signs which had remained essentially the same since the first examination fourteen and a half years earlier. That diagnosis had, in fact, been made after examination at two different hospitals. Dr. Heale pointed out, however, that the signs did not correspond with any of the usually described congenital heart lesions. He said that in congenital subaortic stenosis there was no aortic diastolic murmur, and also aortic regurgitation as a congenital lesion was unknown except in association with coarctation of the aorta; careful examination in the case under discussion revealed no signs of coarctation of the aorta. Dr. Heale suggested the diagnosis of rheumatic heart disease, but pointed out that if such a diagnosis was accepted the patient presented several unusual features. Firstly, the presence of the obvious signs at the age of ten years necessarily placed the original infection some years previously, because such gross structural changes would take at least several years to develop. Furthermore, there were no signs of mitral stenosis, either clinically or radiologically. Finally, it was remarkable that the patient did not show more limitation in the amount of exertion he could undertake. In spite of those points, Dr. Heale thought that the signs were best explained on the basis of rheumatic heart disease.

Raynaud's Phenomenon.

Dr. Heale also showed a male patient, aged thirty-five years, who had attended at Saint Vincent's Hospital in August, 1939, complaining of abdominal discomfort of several years' duration. During the course of the examination he had mentioned that, in the last twelve months, the fingers of both hands became dead and numb and turned a pale bluish colour whenever they were exposed to cold. He had informed Dr. Heale that the hands were affected unequally; all the fingers of the left hand changed colour, but only the second and fifth fingers of the right hand. He had been operating a vibrating pneumatic tool for the past eighteen months. Dr. Heale said that on examination no evidence of occlusive arterial disease was found; during an attack, which he had seen, the affected fingers were cold and of a pale cyanotic colour as far down as the proximal interphalangeal joints.

Dr. Heale said that Raynaud's phenomenon occurring amongst users of vibrating tools was well recognized. The patient shown was a typical example. The fingers did not become discoloured during work, but only on exposure to cold. The hand holding the vibrating end of the tool was affected first and most. The vibrations transmitted to the hands seemed in some way to sensitize the fingers to cold. Treatment consisted in changing the patient's occupation (if possible) and in protecting the hands against the cold.

Pernicious Anæmia Simulating Heart Disease.

Dr. Heale also showed a male patient, aged sixty-eight years, whom he had attended at Saint Vincent's Hospital in June, 1939, and whose health had been failing for the past fifteen months. The chief complaint was increasing shortness of breath on exertion, and his own medical attendant had diagnosed heart disease with auricular fibrillation; but having noted that the patient was anæmic, he had referred him to the hospital for investigation.

As a result of examination the patient was found to be well nourished, obviously anæmic and easily dyspnœic on slight exertion. The heart was not enlarged and no murmurs were audible, but numerous premature beats were present. The electrocardiographic examination revealed no abnormality apart from the numerous premature ventricular beats. The systolic blood pressure was 150 millimetres and the diastolic 80 millimetres of mercury; the blood examination revealed 2,360,000 erythrocytes and 4,400 leucocytes per cubic millimetre; the hæmoglobin value was estimated as 70% and the colour index was 1·5. The blood film was typical of pernicious anæmia, showing considerable anisocytosis and poikilocytosis and a large proportion of macrocytes. On gastric analysis free hydrochloric acid was absent and the total acidity was low. There were no noteworthy neurological signs; and the only other abnormal physical finding was enlargement of the prostate gland.

Dr. Heale said that treatment had consisted of intramuscular injections of "Campolon", five cubic centimetres twice a week for two weeks, then five cubic centimetres once a week for two weeks and thereafter five cubic centimetres every two weeks; at present the patient looked and felt very well. The results of the latest blood examination, carried out three weeks before the meeting, were the following: the erythrocytes numbered 4,200,000, and the leucocytes were 6,800 per cubic millimetre, the hæmoglobin value was 90% and the colour index was 1·07. Apart from slight anisocytosis, no abnormality was seen in the blood film. Dr. Heale pointed out that patients with anæmia were apt to be erroneously diagnosed as suffering from heart disease. He quoted two cases of pernicious anæmia in which anginal pain was the presenting symptom. He also said that patients with chronic heart disease were anæmic at times, and it was important to recognize the anæmia and treat it, because the anæmia constituted an added burden to the already damaged heart.

Gargoylism.

Dr. H. J. Sinn showed a girl, aged three years, as an example of the condition known as gargoylism. At birth it was noticed that she had "smoky eyes" and snuffles, a large head with frontal protuberance, curvature of the spine and clubbed feet of the *talipes calcaneus* variety. She did not sit up till fifteen months of age; a prominent umbilical hernia was present at eighteen months, and she did not walk until two years and eight months of age. Dr. Sinn said that the baby was breast fed for only two weeks and then received an artificial diet without adequate vitamin content; she had whooping-cough at one year and was subjected to adenoidectomy seven months later on account of the nasal discharge. Even at the time of the meeting her speech was limited to a few words intelligible only to the parents; but she was able to let them known when she wished to defæcate or to micturate. She had an older sister and a younger brother who were normal and healthy. There was no consanguinity in the parentage, nor was mental backwardness recorded on either side of the family. Nocturnal enuresis was noted in the history.

Dr. Sinn said that the child's weight was twenty-nine and a quarter pounds and her height thirty-three and a half inches; the circumference of the head was twenty-one inches. He directed attention to the large head with frontal prominences and the grotesque facies. The bridge of the nose was flattened and the nostrils were wide and faced forwards, with snuffling and much dry secretion. The mouth was large, the lips were broad and the corneæ of the eyes were opaque. The abdomen was protuberant; an umbilical hernia was present and the lower margin of the liver extended about three fingers' breadths below the costal margin; but the spleen was not palpable. Kyphoscoliosis was demonstrable and there was widespread limitation of extension at various joints. From the skiagrams Dr. Sinn showed that the pituitary fossa was elongated and deepened and that there were metaphyseal and epiphyseal changes at the extremities of the long bones and characteristic deficiency in the lumbar vertebræ.

Renal Dwarfism.

Dr. Sinn also showed a boy, aged four years and two months, who was the only child of healthy parents. He had developed fairly well until the age of six months, though labour had been induced two weeks before term because of the mother's kidney trouble. The child's birth weight was four pounds and one ounce and he had been fed on breast milk for a few days only. The small stature of the child had not caused undue concern until approximately five weeks before the meeting, when he had suffered painful spasm of the extremities closely

resembling the spasms of tetany. It was stated that he drank more than an average man and passed large quantities of urine voluntarily by day and involuntarily by night. He was symmetrically dwarfed and weighed only sixteen pounds and two ounces; the head circumference was only eighteen and a half inches, though the head seemed to be proportionately large and the fontanelle was not quite closed. The skin was pale and sallow and relatively inelastic. There was a good air entry into the lungs and no adventitious breath sounds and no heart irregularity had been observed. The limbs were very thin; but there was no enlargement of the bones at the epiphyseal ends. The systolic blood pressure was 90 millimetres of mercury. The fat in the stools had been analysed, but nothing unusual had been found. The urine was clear, like water, with an extremely low specific gravity, and the presence of a small amount of albumin had been detected on every occasion; the tests for sugar were always without result. From the skiagrams it was demonstrated that there was general rarefaction of all bones of the extremities, with increased density at the metaphyseal ends of the long bones; the pituitary fossa was within normal limits of size and shape.

Dr. Sinn said that he had made the diagnosis of renal dwarfism provisionally on the history of dwarfism, polyuria and polydipsia with tetany, and on the urinary and radiographic findings. He proposed to carry out further investigations, including excretion and retrograde pyelography, estimations of the serum calcium and plasma phosphorus contents, the blood and urinary concentration of urea and measurement of the alkali reserve.

Lobectomy.

DR. F. J. COLAHAN showed two patients who had been subjected to lobectomy. The first, a female, aged thirty-two years, had had a chronic lung abscess situated in the upper lobe of the right lung for at least two years. Before operation the amount of sputum had been as much as 450 cubic centimetres daily, and she had had repeated hæmoptyses; clubbing of fingers and toes and great emaciation were features. Right upper lobectomy had been performed, the symptoms had disappeared and the patient had gained in weight by two stone in three months.

The second patient, a male, aged seventeen years, had had advanced suppurative bronchiectasis of the lower lobe of the right lung; the diagnosis had been confirmed by means of bronchograms. The sputum amounted to 400 cubic centimetres daily. Right lower lobectomy had been carried out, with relief of symptoms; the patient was a member of a harriers' club and was able to participate in cross-country races of five miles without difficulty.

Thoracoplasty.

Dr. Colahan also showed two patients who had undergone thoracoplasty. The first, a male, aged twenty-two years, had been bedridden for two and a half years and had been treated in a sanatorium for chronic fibrocavernous pulmonary tuberculosis of the right lung; during the illness he had had repeated hæmoptyses. The operation performed was Semb's nine-rib paravertebral thoracoplasty with apicolysis. He had apparently been cured, as he had gained in weight by two stone and was carrying on a laborious occupation.

The second patient, a male, aged thirty-eight years, had had a large tuberculous cavity in the upper lobe of the left lung. Monaldi's thoracoplasty had been performed and had resulted in apparent cure, with disappearance of the cavity; the patient was able to do hard manual work.

Duodenal Diverticulum.

Dr. Colahan also showed a male patient, aged thirty-four years, upon whom he had operated for an unusual type of duodenal diverticulum. The history was typical of peptic ulcer in the duodenum; but the condition was diagnosed pre-operatively by radiological methods as a duodenal diverticulum. At operation a congenital non-rotation of the duodenum was found with a large diverticulum arising from the third part and situated behind the caudate lobe of the liver. Dr. Colahan had excised the diverticulum and the patient's symptoms were relieved.

Carcinoma of the Stomach.

DR. C. H. OSBORN showed a male patient who had suffered from flatulent dyspepsia for many years, and who had been sent to the hospital in May, 1939, by Dr. C. E. Watson. The patient had experienced a certain amount of epigastric pain at intervals, and this had increased in severity during the past six months. Vomiting had been a prominent feature, and in spite of a variety of diets he continued to vomit frequently and had lost a great deal of weight.

On examination he was a pale, emaciated and elderly looking man, the hæmoglobin value of whose blood was 45%. The abdomen was distended in its upper half and X ray investigation in January had revealed the presence of an ulcer crater on the antral portion of the lesser curvature of the stomach, associated with complete gastric retention over a period of eight hours. A preliminary diagnosis of carcinoma of the stomach was made, and a course of pre-operative medication was embarked upon. This entailed gastric lavage, a dietary high in essential vitamin contents and two blood transfusions. In two weeks the patient's general condition had improved considerably, and his hæmoglobin value was 70%. At this stage laparotomy was performed and a carcinoma was discovered at the site where the ulcer crater had been shown radiologically. There were a few enlarged and hard glands in the subpyloric area. An anterior gastro-jejunostomy was performed, the anastomosis being made as high up on the stomach wall as was possible. The abdomen was then closed, and from that time the patient improved rapidly. It was not until six weeks had elapsed that the second stage of his operation could be attempted, because in the interval he had developed a minor wound infection and pleurisy with a small effusion. When, however, it was embarked upon under high spinal anæsthesia, a subtotal gastrectomy was easily performed, section being made through the first part of the duodenum on the distal side, and proximally immediately adjacent to the former anastomotic opening. The greater and lesser omentum were also removed. Dr. Osborn showed the mounted pathological specimen, together with microscopic slides showing the adenocarcinoma; they had been prepared by Dr. A. Brennan.

An uneventful convalescence followed this procedure, and Dr. Osborn pointed out that he presented the case to illustrate the fact that radical excision for carcinoma of the stomach could be accomplished successfully in poor risk patients, provided proper preparation of the patient was undertaken, and that in suitable cases a two-stage operation was done. This was fraught with far less danger than the radical one-stage gastrectomy which was usually employed. Dr. Osborn went on to say that early diagnosis of carcinoma of the stomach was still an unattained ideal; but that if practitioners would regard all instances of disturbed digestion or unexplained lassitude occurring for the first time in persons over forty years of age as potential cases of carcinoma of the stomach, if they would conduct a simple test for occult blood in the fæces after appropriate preparation, and if those cases in which the tests elicited positive results were thoroughly investigated, far more patients would present themselves in a suitable condition for radical operation.

Adenoma of the Stomach.

The next patient shown by Dr. Osborn was a man, aged sixty-seven years, who presented himself in October, 1938, having noticed seven months previously that he had considerable flatulence before breakfast, associated with slight nausea. If a lighter breakfast than usual was taken the nausea and flatulence would disappear during the forenoon. Some months elapsed, during which time there was complete relief of symptoms; then these recurred and in a more exaggerated fashion, with occasional vomiting and

a feeling of slight epigastric distress. There had been a loss of one stone in weight. Occult blood was present in the stools, but apart from this the clinical examination revealed no abnormality. Radiological investigation, carried out by Dr. John O'Sullivan, revealed in a very beautiful manner a rounded circumscribed filling defect in the antral portion of the stomach, suggesting a fungating tumour. There was also radiological evidence of a duodenal ulcer, although there had been no symptoms suggestive of this disease at any time during the patient's life. A gastroscopic examination confirmed the presence of a fleshy tumour at the site previously mentioned, but no pedicle could be seen although adenoma was suspected.

Under high spinal anæsthesia a subtotal gastrectomy was performed, the distal line of section being through the duodenum, distal to the scar of the old duodenal ulcer, and a Pólya-Finsterer type of anastomosis was performed. Photographs of the specimen and microscopic slides were shown.

Double Carcinoma of the Jejunum.

Dr. Osborn then showed a male patient, aged sixty-seven years, who had been referred to hospital in August, 1939. Umbilical pain, coming on about one hour after meals, had been present for some months and the appetite had been failing for the past eight weeks. There was no nausea; but flatulence after meals was a noticeable feature, and relief followed a gurgling sensation in the vicinity of the umbilicus. There had been no hæmatemesis or melena, but the patient had lost about two stone in weight. Abdominal examination revealed slight rigidity of the left upper rectus muscle, and an indefinite sensation of a movable mass to the left of the umbilicus. The fæces gave a strongly positive result to the test for occult blood. Radiological investigation had been carried out by Dr. Patrick, of the Warrnambool Hospital; Dr. Patrick reported that a gas-filled loop of the jejunum was present, showing a fluid level immediately above a slight constriction, and suggested a diagnosis of carcinoma of the jejunum. Gastroscopic examination revealed no abnormality, and the hæmoglobin value was 75%.

At operation, under high spinal anæsthesia, two tumours were discovered in the upper portion of the jejunum about twelve inches apart, one larger than the other and of a constricting type. The proximal tumour was about three feet from the duodeno-jejunal flexure, and there were numerous enlarged and somewhat firm glands in the neighbouring mesentery. A wide resection of the gut was made, including an appropriate section through the mesentery to include the enlarged glands, and an end-to-end anastomosis was performed. Dr. Osborn showed the pathological specimen, together with microscopic slides prepared by Dr. A. J. Brenan. These revealed ulcerating carcinoma of the jejunum.

The patient's convalescence was uninterrupted as far as his abdominal condition was concerned, but unfortunately gangrene developed in the right lower limb; this necessitated mid-thigh amputation two weeks later. This gangrene had supervened upon a gross state of *endarteritis obliterans* of long standing, for when the femoral artery was divided at the operation it was found to be completely occluded by white fibrous tissue. Another small patch of gangrene had developed on the left heel, but this had not advanced, and the patient was now comparatively well. Dr. Osborn went on to say that although carcinoma of the jejunum was not particularly rare, double carcinoma in this situation had been rarely recorded. He referred at some length to the method of intestinal suture in general that he adopted to ensure sound water-tight anastomosis and avoidance of peritoneal soiling.

Facial Surgery.

Fractures.

Dr. Osborn then presented a series of patients illustrating some aspects of facial surgery. The first was a patient illustrating fracture of the alveolar process of the maxilla treated by an arch bar. The value and application of the arch bar were demonstrated. The most recent model designed by Dr. Prytz was shown.

The second patient in the series had a healed double fracture of the mandible, which had been treated by interdental wiring. This patient had also suffered a depressed fracture of the malar bone. Characteristics of this fracture were demonstrated; Dr. Osborn pointed out that the fracture site was most commonly found to be at the malar-maxillary junction. An X ray photograph in a special plane was necessary to show the fracture properly. Failure to reduce the fracture resulted in a most unsightly deformity, and the technique of the elevation of this bone by means of a strong towel forceps was demonstrated.

The third patient in the series had sustained a double fracture of the mandible through both mental foramina. This patient was treated by interdental wiring in occlusion. As she suffered from a natural retrognathism, it was decided later to improve her profile by advancing the chin. Photographs and skiagrams illustrated the former condition in comparison with the patient's present appearance.

The fourth patient had sustained a simple depression of the malar bone.

Cheiloplasty and Rhinoplasty.

Dr. Osborn then showed six patients who had undergone cheiloplasty and rhinoplasty for old hare-lip deformities, and the principles underlying treatment of the lip and nose were demonstrated, together with photographs taken prior to operation. Two patients on whom operation had been carried out for unsightly noses were shown and the technique was described. Dr. Osborn pointed out the great psychological improvement which was noticeable in these patients after the correction of unsightly deformities of the face.

Cleft Palate.

A patient illustrating secondary repair for cleft palate was presented, showing the results to be obtained by operation carried out according to Wardhill's principles and technique.

Skin Grafting.

Dr. Osborn finally showed two patients illustrating skin grafting. In the first case a full thickness graft to the finger was shown, following "Z-plasty" for scar contracture. In the second a pedicle graft had been made to cover an extensive loss of skin and subcutaneous tissue on the forearm.

Traumatic Epilepsy: Resection of Scar.

DR. FRANK MORGAN showed a female patient, aged eighteen years. Four years previously she had sustained a motor car accident, after which she was unconscious for seven days. Two years later she began to suffer from epileptic fits, which gradually increased in intensity and number, so that four years after the accident she would have as many as twenty to thirty fits over a period of two days, and after these series of attacks would not regain normal consciousness until one week had elapsed. Her mother stated that the head and eyes sometimes turned to the left in the attack.

Examination revealed slight indentation of the bone of the right side of the forehead and a longitudinal scar on the right side just behind the hairline, where an operation had been performed at the time of the original accident. There were no physical signs other than these, except that both plantar reflexes were extensor in type following the epileptic attacks. The cerebro-spinal fluid pressure was not raised and the protein content was 20 milligrammes *per centum*. Encephalography revealed no abnormality of the ventricular system. Because of the history of conjugate deviation of the head and eyes to the left during the attacks, and the slight indentation of the skull in the right frontal region and the position of the operative scar there, right frontal osteoplastic

craniotomy was performed. The dura was intact; but beneath there were adhesions between the cortex and the dura on the superior and orbital surfaces of the frontal lobe. The frontal lobe itself was firm "on palpation of the brain needle". The anterior half of the frontal lobe was resected, the frontal horn being widely opened. Examination of the resected portion of lobe revealed a large central scar. No attacks had occurred since the operation, which had been performed eleven months prior to the meeting.

Astrocytoma of the Cerebellum.

Dr. Morgan's second patient was a female, aged seventeen years, who gave a history of headaches and vomiting of one month's duration.

Examination revealed one diopter of papillœdema on the right side; on the left, papillœdema was just appreciable. There were slight paresis of both external recti muscles, motor ataxia of the left upper limb, dysdiadokokinesia of the left side and hypotonia of the left upper limb. The cerebro-spinal fluid was under a pressure of 400 millimetres of water. X ray examination of the skull revealed irregular areas of calcification in the posterior fossa on the left side, and moderate separation of the sutures of the skull.

Cerebellar exploration was performed. This revealed a large tumour in the left cerebellar lobe extending into the fourth ventricle and pressing on the floor of that cavity. The tumour was removed. After its removal the patient suffered from complete right facial paralysis of nuclear type and paralysis of conjugate deviation of the eyes. These palsies disappeared in the course of a few months. The patient had returned to work. The tumour proved to be an astrocytoma.

Suprasellar Cyst.

Dr. Morgan then showed a female patient, aged ten years, who had always been a delicate child and even as a baby was subject to "sick turns". During several winters she had suffered severely from what was thought to be hay fever and nasal catarrh. For some years she had been subject to attacks of somnolence, during which she would go to sleep for two days and could not be roused either to eat or drink. Fever was noticed in the attacks. Two years earlier she had complained of headache, mostly supraorbital and left temporal. Eight months later visual trouble was noticed; when she closed the right eye she was able to see only half a person with the left eye.

On examination she was seen to be four feet one and three-quarter inches in height; her weight was fifty pounds. Bilateral primary optic atrophy was present; examination of the visual fields revealed bitemporal hemianopia. On the left side the hemianopia was to the mid-line, with sparing of the fixation point for 200/2,000 white. On the right side the hemianopic defect was not so complete as on the left, 200/2,000 being seen as far out as 15°. With the left eye the patient was able to count figures at one foot; the visual acuity of the right eye was $^{6}/_{12}$. An X ray examination revealed considerable enlargement of the *sella turcica*, with depression of the floor and tilting backward of the *dorsum sellæ*.

Left transfrontal osteoplastic craniotomy was performed; this disclosed a cystic tumour lying between the optic nerves, partly within and partly above the region of the sella. The cyst contained 10 cubic centimetres of turbid, brownish-coloured fluid, which contained cholesterin crystals. The cyst was completely removed. Histological examination showed the wall of the cyst to consist of fibrous tissue containing areas of xanthoma-like cells and giant cells, with patchy leucocytic infiltration of one or two areas. The patient made a good recovery from the operation. Since its performance five months earlier she had increased in weight by twelve pounds and in height by three-quarters of an inch. There were no longer attacks of headache, fever or insomnia. The visual acuity of the left eye was now $^{6}/_{60}$ and of the right eye $^{6}/_{9}$. The visual fields were slowly expanding.

Pituitary Tumour.

The fourth patient shown by Dr. Morgan was a male, aged fifty years. He gave a history of loss of libido of six years' duration, lassitude of twelve months' duration, diplopia of three months' duration and misty vision of four months' duration. During all his life he had shaved only every second day.

On examination, slight greyness of the optic disks and bitemporal hemianopia were found; visual acuity on the right was $^{6}/_{18}$ partly and on the left $^{6}/_{12}$ partly. Paresis of the third nerve was present. There was a slight increase in the sugar tolerance. X ray examination revealed a considerably enlarged pituitary fossa.

Right frontal osteoplastic craniotomy was performed and the pituitary region was approached by the intradural route beneath the frontal lobe. A large pituitary tumour was partially removed by curettage and sucking. After the operation the patient had a complete third nerve palsy on the left side. The visual fields rapidly expanded so as to become full and the vision in each eye recovered to $^{6}/_{4}$. The ocular motor paralysis disappeared within two and a half months of the operation, except that the left pupil remained slightly larger than the right and was considerably less active than the right when stimulated with light.

Pressure on the Cauda Equina.

Dr. Morgan then showed a male patient, aged fifty-one years, who had suffered an injury to his back two years earlier. He had been tired and weak for twelve months after this, and nine months after the accident began to suffer from pain in the right leg, made worse by coughing and sneezing. The pain gradually became worse and for four more months he had pain radiating down the right leg into the left buttock, sufficiently severe to prevent him from working. Any exertion brought on these pains, and also sitting on the right buttock caused pain to shoot down the right leg. The right leg was colder than the left. For eight months before the operation he noticed that the upper portion of his body tilted to the left side. The operation was performed in September, 1938.

Examination revealed considerable wasting of both buttocks and of the hamstring muscles on the right side, a lumbar scoliosis of severe degree with the concavity to the left, and compensatory thoracic scoliosis with the concavity to the right. Correction of the scoliosis caused severe pain to radiate to the right buttock from the middle of the spine in the lumbar region. There was fibrillation of the hamstring muscles on the right side and of the right glutei. The protein content of the cerebro-spinal fluid was 60 milligrammes *per centum*. Four cubic centimetres of lipiodol were injected between the second and third lumbar spines, and X ray pictures were taken in all positions; but no certain abnormality of the *cauda equina* region was demonstrated.

Lumbro-sacral laminectomy was performed; this revealed a greatly enlarged ligament lying between the lamina of the fifth lumbar vertebra and sacrum, which pressed forward on the *dura mater*, tightening it like a bow. Removal of this ligament resulted in instantaneous relief of the pain, spontaneous correction of the scoliosis and return of the substance of the wasted muscles, and ability to work again.

Dr. Morgan finally showed a male patient, aged forty-seven years. After an operation for removal of the internal cartilage of the right knee this patient had complained for three years of weakness in the right hip, knee and ankle, and of neuralgia of the right leg. Ten weeks before operation, which was in September, 1938, he experienced a severe pain in the right buttock, which he described as being like a kick. Following this severe pain there was a slow radiation of pain down the right leg, which reached the toes in one and a half hours. This was followed by a cramp-like contraction of the right leg accompanied by hyperæsthesia of the back of the right leg and the right buttock.

On examination, there was an area of paræsthesia to pin prick and light touch over the distribution of the

fifth lumbar to the third sacral roots on the right side, and a doubtful area of sensory loss to pin prick and light touch over the distribution of the first sacral root area on the right side. There was an extremely tender spot at the junction of the fifth lumbar vertebra and the sacrum. Tenderness was also present along the whole course of the sciatic nerve from its exit at the sciatic notch as far as the ankle. Considerable wasting of the right *quadriceps femoris*, hamstring and glutei muscles was noted. The right knee jerk was diminished and the ankle jerk was absent. Dilated veins were present in the skin over the upper sacral and lower lumbar portions of the spine. The right foot was colder than the left. The protein content of the cerebro-spinal fluid was 40 milligrammes *per centum*.

Lumbro-sacral laminectomy revealed a firm ligamentous structure between the first sacral vertebra and the fifth lumbar vertebra on the right side, which pressed forward on to the fifth lumbar root and on to the spinal dura adjacent to this. Removal of the structure resulted in instantaneous relief of the pain, and had been followed by return of the substance of the wasted muscles. There still remained an area of sensory loss in the distribution of the first sacral segment.

Contact Therapy.

Dr. John O'Sullivan showed patients suffering from carcinomata of the lip, face and dorsum of the hand treated by contact therapy. He emphasized the superiority of this method of treatment in cases of carcinoma of the back of the hand, and pointed out the great risk of using radium needles in this situation, owing to the fact that necrosis nearly always followed this form of treatment. The advantage of the Chaoul method of treatment was that the dose could be spread over a period and adapted very easily to the needs of the case. Dr. O'Sullivan stressed the importance of avoidance of damage to the tissue bed.

Radiology.

Dr. O'Sullivan then demonstrated a series of skiagrams showing the value of bronchography in the diagnosis of pulmonary disorders. He explained his method of performing bronchography and demonstrated his technique, using his simplified type of introducer for the intratracheal catheter.

Among the cases discussed was that of a patient with a history of cough and hæmoptysis extending over many years. Plain postero-anterior and lateral films showed an area of infiltration in the upper lobe of the right lung, in the centre of which a small dense shadow could be seen. Examination after lipiodol injection revealed bronchiectasis localized to the pectoral and axillary branches of the right upper lobe bronchus. A lobectomy was successfully performed by Dr. F. Colahan. The operation specimen confirmed the bronchographic findings. The dense shadow seen on the film was due to a small pebble, of which there had been no history of aspiration.

Dr. O'Sullivan then referred to a case of lung abscess, localized by bronchographic examination to that portion of the lung supplied by the first dorsal branch of the right lower lobe bronchus. Dr. O'Sullivan mentioned that this was quite a common site for the development of a lung abscess. He emphasized the importance of the bronchographic investigation of the whole bronchial tree as a preliminary to surgical procedures on the chest, as it was useless to perform a lobectomy or pneumonectomy when other lobes or the other side of the chest were involved. This would specially apply to cases of bronchiectasis.

Dr. O'Sullivan then showed bronchograms from several cases of bronchial carcinoma. In one case collapse of the middle lobe of the right lung was seen on the plain films. Examination after lipiodol injection revealed a block at the right middle lobe bronchus. This finding was confirmed by bronchoscopy.

Dr. O'Sullivan emphasized the fact that not in all cases of bronchial carcinoma was there a typical atelectatic collapse of the involved lung segment, and he discussed a case in which there was a diffuse mottling of the upper lobe of the right lung which would be consistent with a tuberculous condition. He emphasized the necessity for repeated sputum tests by smear and culture in these cases; if they were without result, he considered that tuberculosis could be ruled out clinically and another cause for the symptoms would be found. Bronchial carcinoma should always be considered in such chronic non-resolving cases of pulmonary involvement with negative results to sputum tests.

A large abscess cavity was shown in the middle lobe of the right lung; the cavity did not fill up with lipiodol until the patient was turned over on the face. This was due to the fact that the middle lobe bronchus came off on the anterior aspect of the right main bronchus.

Dr. O'Sullivan emphasized the fact that all bronchographic examinations should be carried out under fluoroscopic control, with appropriate posturing of the patient to outline the different portions of the bronchial tree.

Dr. O'Sullivan showed a series of films from a case in which a tooth had been aspirated during dental extraction. The first film showed very clearly the atelectatic and infective change in the lower lobe of the right lung. The tooth was removed bronchoscopically, and after some time the lobe cleared up. The next film showed the appearances two years after the removal of the tooth. Mottling was to be seen in the right lower quadrant of the chest. Lipiodol injection revealed bronchiectatic change in this situation. Dr. O'Sullivan said that this was a common sequel of foreign body associated with infection in the lung.

Dr. O'Sullivan discussed a series of cases illustrating the value of tomography. Several examples of tuberculous cavities, which could not be seen clearly by the ordinary method, were quite easily discerned and localized by the tomographic method. Dr. O'Sullivan explained the type of tomograph used by him. He then discussed a number of films showing the value of the tomographic method in the diagnosis of bronchial carcinoma. On the films shown it was easy to observe the trachea and bronchi. In one case the obstruction was in the right upper lobe bronchus, involving its pectoral and axillary branches. The right apical branch was quite clearly outlined. In another case a narrowing of the right main bronchus in the region of the middle lobe bronchus could be clearly seen on the tomographic films. Dr. O'Sullivan considered that this method would render unnecessary bronchographic examination in certain cases.

Dr. O'Sullivan next discussed a case of fungating tumour of the stomach; the tumour could be well seen by means of the mucous membrane technique. It was subsequently successfully removed by Dr. C. Osborn, and was found histologically to be an adenoma of the stomach. Cases of fungating carcinoma of the fundus of the stomach demonstrated by the air filling of the fundus were also discussed.

The value of the radiological control of the treatment of duodenal ulcers was exemplified by a case of acute ulceration on the anterior and posterior walls of the duodenum. The first "aimed snapshot" made revealed a large ulcer crater on the anterior wall and a small one on the posterior wall. After treatment had been given, the ulcer crater on the posterior wall had disappeared, and that on the anterior wall had greatly diminished. At a still later period, the contour of the anterior wall was quite smooth. Dr. O'Sullivan said that in cases such as this it was comparatively easy to give an opinion as to the presence or absence of ulceration, but in the long-standing case with gross deformity it was not always possible to demonstrate the duodenal ulcer crater, and in such cases the radiologist could only report that the duodenal cap was deformed and could not be definite as to the presence or absence of an ulcer crater.

Dr. O'Sullivan then showed skiagrams from a case of multiple carcinomata of the large bowel; he also showed films revealing inflammatory condition of the caecum and ascending colon, which might be regarded as a regional

ileo-colitis. The condition was apparently a localized typhlitis with the same basis as the localized ileo-colitis. By way of contrast, films from a case of fungating tumour of the caecum were shown. Dr. O'Sullivan emphasized the silent nature of such tumours, which gave rise to symptoms due to blood loss and toxæmia rather than to bowel symptomatology. He said that the fundus region of the stomach was another situation in which careful search should be made by the radiologist in obscure cases of cachexia and anæmia.

Films were also shown illustrating the value of the aimed compression technique in the diagnosis of biliary calculi. Dr. O'Sullivan said that this method of examination consisted in visualization of the gall-bladder on the fluoroscopic screen, and then, by means of compression pads on handles, it was possible to apply graduated compression over the gall-bladder. As instancing the value of this method, Dr. O'Sullivan showed a series of films from a case in which there were symptoms of biliary colic; in routine cholecystographic investigations a good filling of the gall-bladder was demonstrated, but not calculi. By means of the dose compression technique, it was possible to demonstrate a line of swimming multiple biliary calculi. X ray films of the gall-bladder after it had been excised were also shown; the calculi were well seen.

A MEETING of the New South Wales Branch of the British Medical Association was held on September 21, 1939, at Sydney Hospital, Macquarie Street, Sydney. The meeting took the form of a number of clinical demonstrations by members of the honorary staff of the hospital.

Lymphosarcoma.

DR. G. C. WILLCOCKS showed a male patient, aged fifty-two years, a labourer, who had been admitted to hospital on August 30, 1939, complaining of lumps in both groins, present for three months. They had not been tender and had not caused any discomfort, but had been gradually increasing in size, particularly on the left side. He had no other symptoms and had had no previous illnesses of any significance.

On examination, enlarged, soft, discrete glands were found in both groins. Apart from the masses the patient exhibited no abnormal physical signs. Special investigations included a blood count, which gave the following results. The erythrocytes numbered 5,370,000 per cubic millimetre; the hæmoglobin value was 115%, or 16 milligrammes *per centum*; the colour index was 1·08 and the hæmoglobin content of the cells was full; slight anisocytosis was present. The leucocytes numbered 8,250 per cubic millimetre, 72% being neutrophile cells, 2% eosinophile cells, 24% lymphocytes, and 2% monocytes.

The Wassermann and Kahn tests elicited no reaction. An X ray examination of the chest revealed no newgrowths or other abnormality. A gland was removed from the groin for examination. The report was: "Suggestive but not diagnostic of Hodgkin's disease." After this the glands in the left groin increased in size rapidly and became much harder.

Dr. Willcocks said that the interesting aspect of the case was that the soft glands and the biopsy report both favoured a diagnosis of lymphadenoma and that the glands had increased rapidly in size and hardness following biopsy, to such a degree that a diagnosis of lymphosarcoma seemed highly probable. Treatment had been confined to an attempt to control the gland swelling by the administration of six doses of deep X rays to both groins.

Pick's Disease.

Dr. Willcocks next showed a male patient, aged thirty-seven years, a carpenter, who had been admitted to hospital on September 4, 1939. He complained of swelling in the epigastrium of twelve months' duration. An attack of breathlessness associated with œdema of the feet and face, which had begun ten weeks earlier, had lasted for three weeks. He had had an attack of nocturnal dyspnœa at that time. He was tired and easily fatigued. He suffered from palpitation at night and from frequency of micturition. Prior to the onset of illness twelve months earlier he had been in excellent health.

The presenting signs of importance were the following: considerable swelling of the abdomen, enlargement of the liver, œdema of the legs, varicose veins in the legs, ascites, an irregular pulse and auricular fibrillation.

The Wassermann and Kahn tests elicited no reaction. An X ray examination revealed enlargement of the heart shadow, especially the left auricle, a doubtful appearance of the upper lobe of the left lung, possibly tuberculous, and enlargement of the liver shadow. Infiltration of the pericardium with an opaque material, probably calcium, was also revealed. A blood count gave the following information. The erythrocytes numbered 5,200,000 per cubic millimetre, the hæmoglobin value was 88%, or 12·3 milligrammes *per centum*, and the colour index was 0·84; slight anisocytosis and poikilocytosis were present. The leucocytes numbered 8,800, of which 72% were neutrophile cells, 2% were eosinophile cells, 24% were lymphocytes, and 2% were monocytes. An electrocardiogram revealed myocardial degeneration and auricular fibrillation, with left preponderance.

The diagnosis was made by the skiagrams, which revealed typical signs of chronic constrictive pericarditis or Pick's disease. Medical treatment was ineffective.

An operation (cardiolysis) was being considered, to endeavour to relieve pressure on the heart and great veins.

Myxœdema.

DR. E. H. STOKES's first patient was a woman, aged sixty-three years, suffering from myxœdema. She stated that she had enjoyed good health up till 1935, when she noticed that her face had become swollen after a dental extraction. Later she noticed swellings of the hands and feet and her hair had fallen out. Her hearing had been poor during recent months. She had become very despondent in the course of her illness. It was interesting to note that when she was about thirty years of age a goitre had been present.

On examination it was seen that she presented the typical features of myxœdema. She was pale, her hair was sparse, and swelling of the face, the supraclavicular regions and the body were evident. The blood pressure readings were: systolic 180 millimetres of mercury, diastolic 120. The basal metabolic rate was – 23% and the blood serum cholesterol content was 370 milligrammes per 100 cubic centimetres. The serum albumin content was 7% and the serum globulin 3%, with a ratio of 2·3. A blood count revealed a slight degree of hyperchromic anæmia with leucopenia. X ray examination revealed enormous enlargement of the heart shadows, the left ventricle being especially affected. Electrocardiographic examination disclosed low voltage of the *Q R S* complex with poor development of the *T* wave. Thyreoid tablets, one grain, were prescribed, to be taken three times a day after meals. At the time of the meeting the patient's condition had shown considerable improvement. Dr. Stokes remarked that cases of frank myxœdema were uncommon nowadays.

Chronic Pulmonary Abscess.

Dr. Stokes's next patient was a man, aged thirty-eight years, who had suffered from pneumonia affecting the base of the right lung fourteen months previously. Some teeth had been extracted under local anæsthesia four months prior to the onset of his illness. Apparently the pneumonic condition had resolved slowly, and he expectorated large quantities of foul sputum for many months. It was thought at one time that he might be suffering from pulmonary tuberculosis; but numerous examinations of the sputum revealed no tubercle bacilli.

On examination, on June 26, 1939, it was noted that the ribs over the base of the right lung had fallen in. Dulness on percussion was found and crepitations were heard over the lower lobe of the right lung. Radiographic examination revealed elevation and fixation of the right side of the diaphragm, due to thickened pleura and adhesions. No abnormality was demonstrated after injection of lipiodol. The fingers were clubbed.

Dr. Stokes remarked that this patient was making good progress, and it appeared that Nature was endeavouring to obliterate the abscess cavity which apparently had existed earlier in the disease. It was proposed to have a bronchoscopic examination made later, as new growth was sometimes present in such cases.

Carcinoma of the Œsophagus.

Dr. Stokes next showed a woman, aged sixty-seven years, suffering from carcinoma of the œsophagus. Five weeks prior to the meeting she had noticed difficulty in swallowing and she had lost considerable weight.

On examination her dehydrated appearance was at once evident. There was a large, hard, glandular mass above the right clavicle, about one inch lateral to the right sterno-clavicular joint. An X ray examination revealed an obstruction at the level of the body of the third thoracic vertebra just above the aortic arch. It appeared that two inches of the œsophagus were involved. The blood serum failed to react to the Wassermann test. Dr. Stokes pointed out that early involvement of supraclavicular glands was of bad prognostic omen.

A Neurological Case for Diagnosis.

Dr. Stokes then showed a man, aged forty years, who had suffered from weakness and lassitude during the past ten years, from pain, numbness and tingling of both legs for five months, and from failing vision for three months.

On examination, tenderness of the calves of both legs was evident; the knee and ankle jerks were normal; double papilœdema and contraction of the fields of vision were noted. The blood and the cerebro-spinal fluid failed to react to the Wassermann test. Dr. Stokes said that the case presented features suggestive of multiple neuritis. The patient had been given injections of "Vibex" during the past three weeks and had shown some slight improvement.

Carotinæmia.

Dr. Stokes's last patient was a man, aged sixty-five years, who had been suffering from diabetes for several years past. He had eaten large quantities of pumpkin, and early in 1938 the pigmentation characteristic of carotinæmia made its appearance. His diet had been altered and more green vegetables were given to him; the pigmentation had become less pronounced.

Myxœdema with Hyperchromic Anæmia.

DR. K. B. NOAD showed twin sisters, aged forty-six years, suffering from myxœdema associated with hyperchromic anæmia. He said that they illustrated the simple hyperchromic type of anæmia which occurred in myxœdema, as distinct from the Addisonian hyperchromic anæmia which might also complicate the disorder. The characteristics of the former were that anæmia was never severe, that macrocytosis was present without excessive anisocytosis, and that very little evidence of blood regeneration was seen. Gastric acidity might be normal or achlorhydria might be present. The anæmia disappeared slowly with thyreoid therapy alone. Addisonian hyperchromic anæmia had all the usual features of that disease—macrocytosis with excessive anisocytosis and signs of blood regeneration, achylia, characteristic tongue and possibly cord signs. Liver therapy was required in addition to thyreoid. The common type of anæmia in myxœdema was of the hypochromic variety; but the occurrence of hyperchromic anæmia should not be forgotten. The simple hyperchromic form was the result of an atrophy of the erythron in response to lessened oxygen requirements of the patient. The occurrence of myxœdema and that type of anæmia in twins was particularly interesting.

Dr. Noad said that the first twin had attended the out-patient department on December 2, 1938. She complained of always being cold, of dryness of the skin, of breathlessness on exertion and of pain in the lower part of her back. She had never had dyspepsia or any bowel difficulty. Her menstrual periods were regular and normal. Her previous illnesses had consisted of measles and mumps only. The two sisters were born in England and had come to Australia eighteen years earlier.

Physical examination revealed pronounced myxœdema and pallor of the mucous membranes. No abnormality was found in any other system. The basal metabolic rate was – 27%. Examination after a test meal revealed complete achlorhydria. The blood cholesterol content was 122 milligrammes *per centum* after treatment. A blood count gave the following information: the erythrocytes numbered 2,990,000 per cubic millimetre, the hæmoglobin value was 61% and the colour index was 1·16. The red cells were full of hæmoglobin. Moderate anisocytosis and slight poikilocytosis were noted. The leucocytes numbered 5,520 per cubic millimetre; 76% were neutrophile cells, 16% were lymphocytes and 8% were monocytes. Of the neutrophile cells 2% were band forms.

The patient was given thyreoid tablets, one grain, in increasing doses till five grains were being given each day. She later received parenteral injections of a crude liver extract preparation, of "Campolon" and later of P.D. extract. She was also given a saturated solution of *Ferri et Ammonii Citras*, one drachm twice a day.

The patient's progress was revealed by successive blood counts. The first revealed 3,450,000 erythrocytes and 3,450 leucocytes per cubic millimetre, a hæmoglobin value of 67% and a colour index of 0·95. The second revealed 4,090,000 erythrocytes per cubic millimetre, a hæmoglobin value of 73% and a colour index of 0·91. The erythrocytes were full and slight anisocytosis was present. The leucocytes numbered 6,160 per cubic millimetre. All cells were mature. The third blood count revealed that the erythrocytes numbered 4,080,000 per cubic millimetre, the hæmoglobin value was 81% and the colour index was 1·0. The erythrocytes were full and slight anisocytosis was present. The leucocytes numbered 4,300 per cubic millimetre; 88% were neutrophile cells, 11% were lymphocytes and 1% were monocytes.

The second twin, shown by courtesy of Dr. J. H. Cramsie, said that she was "born rickety and without any blood". She did not walk till the age of four years. Eleven years earlier she had an attack of sciatica on the right side, which had lasted for six weeks. She had never had much energy, but her health was fairly good until two years prior to the meeting, when pain returned in the right leg. It extended from the buttock to the toes and ached constantly. She endeavoured to carry on for one year, but it was then so bad that she had to seek medical advice, and she had been in bed practically ever since. She felt cold constantly, and used to wear "two of everything". She always had a feeling of "cold water on the tops of the legs". Her skin was dry, and she became slow in thinking and her memory was bad. Indigestion had not troubled her and her bowel function had been satisfactory. Menstruation had been regular; the menstrual loss was profuse before her present treatment, but had lessened since.

Several special investigations were made. No record was available of the basal metbolic rate at the height of the myxœdema. On September 21, 1939, it was + 12%. The blood cholesterol content was 189 milligrammes *per centum* after treatment. A blood count made in 1938 gave the following information: the erythrocytes numbered 2,970,000 per cubic millimetre, the hæmoglobin value was 66% and the colour index was 1·13. Moderate anisocytosis was present. Macrocytes were numerous and an occasional microcyte was seen. Slight poikilocytosis was noted. No nucleated erythrocytes were seen. The leucocytes numbered 10,800 per cubic millimetre; 85% were neutrophile cells, 9% were lymphocytes, 4% were monocytes and 2% were eosinophile cells.

Another blood count on September 21, 1939, gave the following information: the erythrocytes numbered 3,320,000 per cubic millimetre, the hæmoglobin value was 74% and the colour index was 1·1. The cells were full of hæmoglobin. Slight anisocytosis was present. The leucocytes numbered 3,800 per cubic millimetre; 71% were neutrophile cells, 4% were eosinophile cells, 22% were lymphocytes and 3% were monocytes. Platelets were plentiful.

The gastric acidity could not be determined, as the patient was unable to swallow the tube.

Since June, 1939, the patient had been receiving parenteral injections of liver. She had been taking nine grains of thyreoid extract a day since July, 1939.

Thrombosis of Posterior Inferior Cerebellar Artery.

Dr. Noad then showed a male patient, aged sixty-two years, a brick carter. He complained of the sudden onset of inability to swallow at breakfast one morning. Intense giddiness followed; he became so giddy that he fell to the ground, but he did not lose consciousness. He was nauseated, but did not vomit, and his vision was blurred. In the succeeding days he was unable to speak distinctly and slurred his words, and food regurgitated through his nose. At no time was there any loss of power in the limbs.

The dysphagia and dysarthria had decreased. The pupils were small and slightly irregular, and reacted to light through a small range. Ocular movements were full and equal. Nystagmus was present when the patient looked to either side. There was paralysis of the right side of the soft palate and the right vocal cord. The gait was ataxic. Slight ataxia was revealed in the finger-nose test. There was complete left hemianæsthesia of the dissociated type—pain, heat and cold were affected, but touch and proprioceptive sensibility were retained. The Wassermann test elicited an incomplete positive reaction. The Kahn test elicited a strongly positive reaction.

Dr. Noad said that this case illustrated a variation of the syndrome, in that the descending root of the fifth nerve on the affected side had escaped and a hemianæsthesia of dissociated type was present, due to involvement of the spino-bulbo-thalamic tract. Considerable variation in the arterial supply of this area normally existed. Another unusual feature was the fact that the plantar reflex was extensor in type on the contralateral side. Dr. Noad thought that it might be due to an extension forward of the area involved, with damage to the pyramidal tract.

Urological Conditions.

DR. REGINALD BRIDGE and DR. ARTHUR CARRODUS showed several patients with urological disorders. The first patient was a male, aged forty-three years, who had attended the out-patient department complaining of hæmaturia of one month's duration. Cystoscopic examination revealed what was apparently a large carcinoma of the vertex of the bladder, admirably suited to partial cystectomy. The patient was admitted to hospital and the tumour was removed. It was circular, 4·5 centimetres in diameter, and resembled a carcinoma, being covered with bullæ and necrotic on the summit, apparently about to fungate. On section, the cut surface resembled a uterine fibroid growth. Microscopically the tumour was found to be a degenerating leiomyoma. The patient was now doing well and the prognosis was good.

The second patient was a male, aged sixty-five years, who had been admitted to hospital with the provisional diagnosis of hypertension and early uræmia. At the time he was vomiting. He was referred to the urologists for examination. A urethral stricture was dilated and a catheter was inserted. Thick viscid urine oozed out of the catheter. A cystogram was made and revealed two large diverticula. After one month's catheter drainage of the bladder the patient's general condition improved considerably and the blood urea level fell to normal. A cystoscopic examination was then performed, and revealed cystitis and considerable trabeculation of the bladder, with many cellules and two diverticula. A median bar was present and was regarded as an important factor in causing the vesical changes. The following day the median bar was resected through an external urethrotomy incision because of the urethral stricture. The patient was now very well and passed urine twice at night without difficulty. The urine still contained pus when microscopically examined. Dr. Bridge and Dr. Carrodus remarked that this case was interesting, in that no calculi were present in spite of the residual infected urine.

The third patient was a male, aged seventeen years, who had been admitted to hospital complaining of frequency of micturition and dysuria of three months' duration. He had had osteomyelitis of the ilium four years earlier.

On examination, a tender mass was palpable in the left iliac fossa. The prostate gland was considerably enlarged, firm and tender. The condition was regarded as possibly periprostatic suppuration following osteomyelitis, or prostatic sarcoma. Radiographic examination revealed low-grade osteomyelitis of the pubic bone. A fortnight later a hard, tender swelling was palpable above the *symphysis pubis*. This was explored and much pus was evacuated from the cave of Retzius. This was drained and the patient made an uneventful recovery. A skiagram showed the bone infection to be healed.

Dr. Bridge and Dr. Carrodus finally showed a male patient, aged sixty-eight years, who had been admitted to hospital complaining of retention of urine of twenty-four hours' duration. He had had gonorrhœa when twenty years of age, but had received no treatment. During the past twenty years he had noticed gradually increasing dysuria and had had two previous attacks of retention of urine, which were spontaneously relieved. The prostate gland was slightly enlarged. The figures arrived at on estimation of the urea nitrogen and creatinine contents of the blood were 70 and 3 milligrammes *per centum* respectively. Suprapubic cystostomy was performed. Ten days later the figures were respectively 30 and 1·5. External urethrotomy was performed and a catheter was inserted. After the removal of the catheter the suprapubic wound, which had healed, broke down and discharged urine. A vesical neck obstruction was suspected, and cystoscopic examination revealed enlarged lateral lobes of the prostate. The adenoma was removed suprapubicly. The suprapubic wound had now healed and the patient had no frequency of micturition or dysuria.

Dr. Bridge and Dr. Carrodus also showed skiagrams and pathological specimens of urological interest.

(To be continued.)

VICTORIAN BRANCH NEWS.

THE following recent judgement of the Workers' Compensation Board of Victoria in the case Stephenson *versus* Victorian Railways Commissioners (3477/39) is published at the request of the Council of the Victorian Branch of the British Medical Association for the information of members.

Stephenson v. Victorian Railways Commissioners.

The Applicant, George Elmslie Leopold Stephenson, has made a claim for compensation upon the ground that on the 15th day of September 1939 and/or on the 18th day of September 1939 he sustained personal injury by accident arising out of and in the course of his employment by the Respondent. The injury of which he complains is a direct inguinal hernia on the right side.

According to the evidence of the Applicant, which was not disputed and which we accept, it appears that on Friday the 15th day of September last in the course of his duties as a goods guard at about 6.45 p.m. at Parwan Railway Station he was required to release an automatic coupling which was stiff and the handle of which was about 3′ 6″ from the ground. In order to release the coupling he gave the handle an extra hard tug and immediately thereafter felt a pain in his right side, as he says, right up the right side. He grabbed his side and doubled up. He told two of his fellow workers what had happened to him and did not complete the work of releasing the coupling. He did no further work that evening and later returned to North Melbourne.

On his journey to North Melbourne he felt as if someone had put a knife in his side and ran it along. He reported his condition to one Stevenson and signed off at 9.14 p.m.

When he reported to Stevenson, the Applicant, according to Stevenson was hunched up and his face was drawn and he was holding his hand on his groin.

On the two following days—Saturday and Sunday—he had no work to do, but early on Monday morning he had to take the 2.45 a.m. train to Geelong and in the course of the journey at Laverton Railway Station he was engaged in putting off trucks and discharging van goods and while shifting a case weighing about 2 cwt he felt a pain go up his side.

He said as he was walking back to the van he felt as if his inside was coming down. He also felt very sick and lay down on the floor of the van. He did thereafter only what he had to do, finding it very difficult so to do and having from time to time to hold his side owing to the pain.

At Werribee he took over a goods train from Geelong and arrived back in Melbourne at 10.25 p.m. and then went home. When undressing that evening he noticed for the first time a lump about the size of a pigeon's egg in his groin. He worked the following day and when he finished the lump was still there and he thereupon consulted Dr. Shannon. Dr. Shannon found that he had a hernia in the right side about the size of half a walnut and he said that after operating on him on the 25th day of September 1939 he discovered a direct inguinal hernia on the right side.

The Applicant never before had any tenderness, soreness or lump in the region of the groin.

The Applicant did not return to his work after the 20th September and was passed as fit for work on the 14th December.

The Applicant on these facts claims that the direct inguinal hernia from which he suffered was an injury arising out of and in the course of his employment.

The Respondent says that the Applicant had a right inguinal hernia for some time prior to the injury alleged and that the work he was performing at the material time did not contribute in any material degree or at all to the occurrence of the hernia. It was also contended that the Applicant's claim was concluded in favour of the Respondent by the decision of this Board in *Bruce v Shire of Hampden*.

The question which we have to decide in this case is ultimately a question of fact, to be determined upon the evidence adduced before us.

There are certain outstanding facts on this case which are not and perhaps cannot be contested. The Applicant whilst exerting some physical force on the 15th September in trying to release an automatic coupling felt a severe pain in his right side and was unable to continue his work for the remainder of that day. On the 18th September, while attempting to shift a heavy case he again experienced great pain in his right side and suffered a feeling of sickness and on the evening of that day noticed for the first time a lump in the groin, and shortly thereafter he is found to be suffering from a direct inguinal hernia in his right side. And the Applicant never before had had any tenderness, soreness or lump in the groin.

Having carefully considered the evidence called for the Applicant and the Respondent we think it proper to conclude that the pain and sickness from which the Applicant suffered in the course of his work on the 15th and 18th days of September 1939 and the appearance of the hernia cannot be attributed to coincidence, and we consider that in this case we are fully justified in applying the following dictum of Lord Birkenhead in the case of Lancaster v Blackwell Colliery Co. Ltd. 12 B.W.C.C. 400 at 412:

"But where the known facts are not equally consistent, where there is ground for comparing and balancing probabilities as to their respective value and where a reasonable man might hold that the more probable conclusion is that for which the Applicant contends then the Arbitrator is justified in drawing an inference in his favour."

The congenital theory of hernia referred to in the case of *Bruce v Shire of Hampden* and discussed before us is not concerned with the direct inguinal hernia from which the Applicant suffered and upon this theory it is unnecessary in this case to express any opinion.

We consider having regard to the evidence of Sir Alan Newton and Mr. Doyle that a hernia may occur without a lump being immediately detected, and that when a hernia does occur it may not necessarily be accompanied by an incapacitating pain.

Sir Alan Newton said that even where a person has a congenital predisposition to hernia the hernia is due to excessive strain and according to Mr. Doyle one can always search and find a cause of a direct hernia. Mr. Doyle further said that the condition of the Applicant which he had heard described would be materially contributed to by the pulling effort which the Applicant himself described.

Having regard to this evidence and to the evidence of the Applicant we are unable to adopt the view expressed by Mr. Trinca that the Applicant's injury was due to a strain of the superficial abdominal muscles and that the pain from which the Applicant suffered was an indication of muscular strain and not of hernia.

In our opinion the direct inguinal hernia from which the Applicant suffered was caused or materially contributed to by the work in which he was engaged on the 15th and 18th days of September 1939, and we find that this injury was caused by accident arising out of and in the course of his employment by the Respondent.

We also find that as a result of this injury the Applicant was totally incapacitated for work from the 20th September 1939 until the 14th day of December 1939. When Dr. Shannon saw the Applicant he considered that he should be operated on and Mr. Trinca said that in his opinion an operation was reasonable and necessary.

There is no dispute about the rate of compensation payable to the Applicant and we therefore award that the Respondent pay to the Applicant as compensation in respect of the injury sustained by him the sum of £30, being compensation at the rate of £2 10s. a week for a period of 12 weeks.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 34, of February 22, 1940.

AUSTRALIAN MILITARY FORCES.

SOUTHERN COMMAND.

Third Military District.

Australian Army Medical Corps.

To be Captain (provisionally) supernumerary to establishment pending absorption—Robert Henry Levinge Dunn, 23rd January, 1940.

Australian Army Medical Corps Reserve.

To be Honorary Captains—Leslie Frank Nicholson, Donald Pryde, William Roy Angus, Eril Julian Larré Claridge, Frederick George Middleton, Charles Edward Stewart, Peter James Parsons, William Alfred Eric Robertson, Wilfred Ewart Macmillan, John Richard Stawell, Horace Finn Tucker, Raymond Paul Gurry, Edward Ernest Joseph Spring, Alister Ashworth Hinchley, Edward Clifford Varley, James Gerald Roy Felstead, Robert Leo Fulton, Kenneth Latham Langlands, William Joseph Rawlings, Gwilym Taliesin James, Colin Wallace Ross and Leslie James Middleton, 22nd January, 1940.

WESTERN COMMAND.

Fifth Military District.

Australian Army Medical Corps.

To be Major (temporarily)—Captain H. M. Trethowan and to remain seconded, 10th October, 1939; Captain

H. G. D. Breidahl and Captain (provisionally) W. Cawley to be brought on the authorized establishment, 15th January, 1940.

Australian Army Medical Corps Reserve.

To be Honorary Captains—Arthur Gordon Abbott, 18th January, 1940, Harry Campbell Pope and Lindsay Poplin Gray, 3rd February, 1940.

Analytical Department.

"STERELIN."

THE antiseptic qualities of melaleuca oil are well known to members of the medical profession. "Sterelin" is an antiseptic made from this oil. It is put up by the Melaleuca Laboratories, of 24, Bond Street, Sydney. The manufacturers claim that it is non-irritant and non-poisonous and may be used for the dressing of wounds, or as a gargle, mouth wash or douche. A solution of 2·5% is said to kill *Bacillus typhosus* in sixty seconds. A sample of this antiseptic was sent to our bacteriologist, who found that the claims of the manufacturers could be substantiated. It may be recommended for the purposes mentioned above.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

THE degree of Doctor of Medicine (M.D.) has been conferred upon Douglas Joseph Anderson.

The Senate has fixed the date for the ceremony of matriculation for Thursday, April 4, 1940.

University lectures will definitely begin on Wednesday, March 27, at 9 o'clock. This applies to all faculties and departments of the university except the senior years in medicine and second-year pharmacy students.

The ceremony of the conferring of degrees will be held later this year on account of the holding of special deferred examinations during the month of April. Degrees in arts and law will be conferred on Saturday, June 15; those in other faculties will be conferred on Saturday, June 22.

Mr. F. Chong, M.Sc. (Sydney) and B.A. (Cambridge), has been appointed assistant lecturer in mathematics and physics at the New England University College, Armidale. Mr. Chong was born in Sydney in March, 1915. He graduated bachelor of science in 1935 with first-class honours and the university medal in mathematics and distinction in physics III. He was admitted to the degree of master of science in 1937. As the holder of the Barker Graduate Scholarship for Mathematics, he proceeded to Cambridge, where he graduated in 1939, being wrangler at the mathematical tripos and gaining honours in part III (topology, geometry, theory of groups and relativity). Mr. Chong was acting assistant lecturer in mathematics at Sydney in 1936 and he has acted as a part-time lecturer in physics.

The position of post-graduate lecturer in pathology and director of the pathological unit at the Prince Henry Hospital has been filled by the appointment of Dr. Frank Byrom, of the pathological institute of London Hospital.

Changes in the Curriculum of the First, Second and Third Years in the Faculty of Medicine.

The following alterations have been approved by the Senate:

First year.—The courses in chemistry I and physics I to be unchanged for the present. The courses in botany I and zoology I to be reduced from 212 to 175 hours. A course in anatomy of approximately 200 hours throughout the three terms to be instituted and an examination in anatomy to be held at the end of the year.

Second year.—Anatomy to extend over three terms, with the final examination at the end of the year. The physiology course to remain as at present. A preliminary course in biochemistry to be given.

Third year.—Physiology and biochemistry and applied anatomy to be given during the first and second terms, the final examinations to be completed by the end of Trinity term and the deferred examinations to be completed and the results published by the end of September.

The foregoing changes will be brought into operation immediately.

Notice.

CLINICAL LECTURES IN SYDNEY.

THE Executive Committee of the Medical Board of the Royal Prince Alfred Hospital has decided that the clinical section of the monthly staff meetings shall be open for attendance to any member of the British Medical Association. The next meeting will be held in the A2 lecture theatre on Wednesday, March 13, 1940. The clinical section meeting will last approximately one hour and will commence at 4.30 p.m. sharp.

A detailed programme of the cases to be presented is as follows:

1. Demonstration of a case of thrombocytopenic purpura and the result of splenectomy, by Professor C. G. Lambie.
2. Clinico-pathological demonstration of malignant tumours of obscure origin, with large secondary deposits beneath the skin, by Professor C. G. Lambie and Dr. G. Davies.
3. Demonstration of a case of typhoid fever with multiple relapses, by Professor C. G. Lambie.
4. Demonstration of a case of pyrexia of obscure origin, by Dr. Kempson Maddox.
5. Demonstration of a case of myxœdema with electrocardiograms in myxœdema, by Dr. Kempson Maddox.

Corrigendum.

THE AUSTRALIAN ORTHOPÆDIC ASSOCIATION.

BY an unfortunate error a footnote was appended to the paper by Dr. Thomas King on the value of femoral osteotomy for diseases and injuries of the hip joint, indicating that the Australian Orthopædic Association is connected with the British Medical Association. The Australian Orthopædic Association is what we have called an extramural association and is in no way part of the organization of the British Medical Association.

Obituary.

JAMES ADDINGTON CALDWELL.

WE regret to announce the death of Dr. James Addington Caldwell, which occurred on February 25, 1940, at Stanmore, New South Wales.

CECIL DANFORD GREENWOOD.

WE regret to announce the death of Dr. Cecil Danford Greenwood, which occurred on February 28, 1940, at Sydney, New South Wales.

Books Received.

TUBERCULOSIS OF THE UPPER RESPIRATORY TRACTS, by F. C. Ormerod, M.D., F.R.C.S.; 1939. London: John Bale Medical Publications Limited. Demy 8vo, pp. 223, with illustrations. Price: 21s. net.

ASTHMA AND THE GENERAL PRACTITIONER, by J. Adam, M.A., M.D., F.R.F.P.S.G., with a foreword by J. Bridie, M.D.; 1939. London: Baillière, Tindall and Cox. Demy 8vo, pp. 168. Price: 6s. net.

LEAGUE OF NATIONS PUBLICATIONS. ENQUIRY INTO MEASURES OF REHABILITATION OF PROSTITUTES, PART III AND PART IV: METHODS OF REHABILITATION OF ADULT PROSTITUTES; CONCLUSIONS AND RECOMMENDATIONS; 1939. Geneva: League of Nations Publications Department; Australia: H. A. Goddard. Demy 8vo, pp. 157. Price: 3s. 6d. net.

VADE MECUM OF MEDICAL TREATMENT, by W. G. Sears, M.D., M.R.C.P.; Second Edition; 1939. London: Edward Arnold and Company. Crown 8vo, pp. 383. Price: 10s. 6d. net.

A SYMPOSIUM ON THE BLOOD AND BLOOD-FORMING ORGANS, by E. Meulengracht *et alii*; 1939. Madison: University of Wisconsin Press. Medium 8vo, pp. 372, with illustrations. Price: $3.50 net.

PHYSIOLOGICAL CHEMISTRY: A TEXT-BOOK FOR STUDENTS, by A. P. Mathews, Ph.D.; Sixth Edition; 1939. London: Baillière, Tindall and Cox. Medium 8vo, pp. 1505, with illustrations. Price: 44s. net.

A NEW DICTIONARY FOR NURSES, compiled by L. Oakes, S.R.N., D.N., assisted by T. B. Davie, B.A., M.D., M.R.C.P.; Sixth Edition; 1940. Edinburgh: E. and S. Livingstone. Crown 16mo, pp. 410, with illustrations. Price: 3s. net.

AN ATLAS OF THE COMMONER SKIN DISEASES, WITH 120 PLATES REPRODUCED BY DIRECT COLOUR PHOTOGRAPHY FROM THE LIVING SUBJECT, by H. C. G. Semon, M.A., D.M., F.R.C.P., photography under the direction of A. Moritz, B.A., M.B., B.C.; Second Edition; 1940. Bristol: John Wright and Sons Limited. Crown 4to, pp. 284. Price: 42s. net.

A SYNOPSIS OF SURGERY, by E. W. Hey Groves, M.S., M.D., B.Sc., F.R.C.S.; Eleventh Edition; 1940. Bristol: John Wright and Sons Limited. Crown 8vo, pp. 722, with illustrations. Price: 17s. 6d. net.

Diary for the Month.

MAR. 12.—Tasmanian Branch, B.M.A.: Branch.
MAR. 12.—New South Wales Branch, B.M.A.: Ethics Committee; Executive and Finance Committee.
MAR. 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAR. 20.—Western Australian Branch, B.M.A.: Branch.
MAR. 26.—New South Wales Branch, B.M.A.: Council (Quarterly).
MAR. 27.—Victorian Branch, B.M.A.: Council.
MAR. 28.—New South Wales Branch, B.M.A.: Annual Meeting.
MAR. 28.—South Australian Branch, B.M.A.: Branch.
MAR. 29.—Queensland Branch, B.M.A.: Council.
APR. 2.—New South Wales Branch, B.M.A.: Council.
APR. 3.—Victorian Branch, B.M.A.: Branch.
APR. 3.—Western Australian Branch, B.M.A.: Council.
APR. 4.—South Australian Branch, B.M.A.: Council.
APR. 5.—Queensland Branch, B.M.A.: Branch.
APR. 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee; Organization and Science Committee.
APR. 9.—Tasmanian Branch, B.M.A.: Branch.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi-xix.

AYR HOSPITAL BOARD, AYR, QUEENSLAND: Resident Medical Officer.

DEPARTMENT OF PUBLIC HEALTH, SYDNEY, NEW SOUTH WALES: Medical Officers.

LIDCOMBE STATE HOSPITAL, LIDCOMBE, NEW SOUTH WALES: Honorary Ophthalmic Surgeon.

ROCKHAMPTON HOSPITALS BOARD, ROCKHAMPTON, QUEENSLAND: Assistant Medical Superintendent.

ST. GEORGE DISTRICT HOSPITAL, KOGARAH, NEW SOUTH WALES: Senior Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	Associated Medical Services Limited. All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Federated Mutual Medical Benefit Society. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	Wiluna Hospital. All Contract Practice Appointments in Western Australia.

Editorial Notices.

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